

Name: _____



2010 CA-1 TUTORIAL TEXTBOOK

4th Edition

STANFORD UNIVERSITY
MEDICAL CENTER
DEPARTMENT OF ANESTHESIOLOGY

Aileen Adriano, M.D.
Kate Ellerbrock, M.D.
Becky Wong, M.D.



TABLE OF CONTENTS

Introduction.....	ii
Acknowledgements.....	iii
Contributors.....	iv
Key Points and Expectations.....	v
Goals of the CA-1 Tutorial Month.....	vi
Checklist for CA-1 Mentorship Intraoperative Didactics.....	vii
CA-1 Mentorship Intraoperative Didactic Lectures	
Standard Monitors.....	1
Inhalational Agents.....	4
MAC and Awareness.....	7
IV Induction Agents.....	10
Rational Opioid Use.....	13
Intraoperative Hypotension & Hypertension.....	16
Neuromuscular Blocking Agents.....	19
Difficult Airway Algorithm.....	23
Fluid Management	27
Transfusion Therapy.....	31
Hypoxemia.....	35
Electrolyte Abnormalities.....	39
Hypothermia & Shivering.....	44
PONV.....	47
Extubation Criteria & Delayed Emergence.....	50
Laryngospasm & Aspiration.....	53
Oxygen Failure in the OR.....	56
Anaphylaxis.....	59
ACLS.....	62
Malignant Hyperthermia.....	65
Perioperative Antibiotics.....	69
Cognitive Aids Reference Slides.....	72

INTRODUCTION TO THE CA-1 TUTORIAL MONTH

We want to welcome you as the newest members of the Department of Anesthesia at Stanford! Your first weeks and months as an anesthesia resident are exciting, challenging, stressful, and rewarding. Regardless how much or how little experience you have in the field of anesthesiology, the learning curve for the next few months will be very steep. In addition to structured lectures and independent study, you will be primarily responsible for patients as they undergo anesthesia and surgery.

Several years ago, before the development of this mentoring and tutorial system, CA-1's had little structure to their first month. While there were regular intra-operative and didactic lectures, the nuts and bolts of anesthesiology were taught with little continuity. CA-1's worked with different attendings every day and spent as much time adjusting to their particular styles as they did learning the basics of anesthesia practice. Starting in 2007, the first month of residency was overhauled to include mentors: each CA-1 at Stanford was matched with an attending or senior resident for a week at a time. In addition, a tutorial curriculum was refined to give structure to the intra-operative teaching and avoid redundancy in lectures. By all accounts, the system has been a great success!

There is so much material to cover in your first couple months of residency that independent study is a must. Teaching in the OR is lost without a foundation of knowledge. Afternoon lectures are more meaningful if you have already read or discussed the material. This booklet serves as a launching point for independent study. While you review the tutorial with your mentor, use each lecture as a starting point for conversation or questions.

During your mentorship, we hope you can use your mentor as a role model for interacting with patients, surgeons, consultants, nurses and other OR personnel. This month, you will interact with most surgical specialties as well as nurses in the OR, PACU, and ICU. We suggest you introduce yourself to them and draw on their expertise as well.

Nobody expects you to be an independent anesthesia resident after one month of training. You will spend the next three-plus years at Stanford learning the finer points of anesthesia practice, subspecialty anesthesiology, ICU care, pre-operative and post-operative evaluation and management, etc. By the end of this month, we hope you attain a basic knowledge and skill-set that will allow you to understand your environment, know when to ask for help, and determine how to direct self-study. Sprinkled throughout this book, you'll find some light-hearted resident anecdotes from all the good times you'll soon have, too.

Any resident or attending in the department is available for questions or advice. If you have any questions about the mentor program, booklet, or lectures, please direct them to one of us: Becky Wong, Katie Ellerbrock, or Dr. Adriano.

CA-1 Introduction to Anesthesia Lecture Series:

The Introduction to Anesthesia Lecture series is given by attendings designed to introduce you to the basic concepts of anesthesia. Topics covered include basic pharmacology of anesthetics, basic physiology, and various clinical skills and topics. This lecture series starts on Wed, July 7 at 4pm in the Anesthesia Conference room. There will be lecture on Thurs, July 8, and then

every subsequent Tuesday, Wednesday, and Thursday at 4pm in July. The last lecture is July 29th. You will be relieved of all clinical duties to attend these lectures. The department has purchased Miller's *Basics of Anesthesia* for use as a reference for these lectures. Dr. Jaffe's book *Anesthesiologist's Manual of Surgical Procedures* is an invaluable resource for understanding the surgical aspects of your anesthetic.

ACKNOWLEDGEMENTS

We would like to thank Dr. Adriano for being the faculty director of the CA-1 Mentor Program. She is a very enthusiastic teacher and knowledgeable anesthesiologist. Despite being a new mom, she has worked tirelessly to prepare for your arrival, organizing this mentorship program and lecture series months in advance. Over the next year, you will likely have the opportunity to work with her one on one in the OR. Maybe she'll even show you pictures of her new baby!

Dr. Harrison will be the advisor for the Class of 2013. He is a graduate of the Stanford Anesthesiology Residency, and you'll find him at the VA doing general and cardiac cases. He is also actively involved in the simulator sessions which you will have the opportunity to do once at the beginning of your CA1 year, again later in the CA1 year, and then once every year thereafter. He's a great teacher and passionate about resident education.

Thanks to Dr. Goldhaber-Fiebert (she'll probably have you call her "Sara") for her dedication to developing and improving the cognitive aids you'll see in this book and in the laminated cards you'll receive. She loves to teach and is good at it, as you'll soon see in the Stanford ORs and the VA simulator sessions. Also thanks to Kam McCowan for her work with Dr. Goldhaber-Fiebert with the cognitive aids.

Thanks to Janine Roberts for her hard work and assistance in constructing the CA-1 Mentorship Textbook, as well as her instrumental role in coordinating the CA-1 Introductory Lecture Series. If you haven't already noticed, she has the answer to nearly everything.

Thanks to Dr. Pearl for his support and assistance with this endeavor. His guidance is appreciated by all. If you ever feel like you're staying too late, know that Dr. Pearl is probably still working in his office when you leave the OR.

Thanks to Dr. Macario, Residency Program Director, who will be one of the first attendings to know all of you by your first names.

Special thanks to Dr. Ryan Green, Class of 2008, founder of the CA-1 mentorship program, and principal editor of the first edition of the CA-1 Mentorship Textbook.

Lastly, thanks to all of the resident and faculty mentors at Stanford University Medical Center, Palo Alto VA, and Santa Clara Valley Medical Center for all of their time and effort spent teaching our program's residents.

Welcome to Stanford Anesthesia.
We hope you love it as much as we do!

Becky Wong and Kate Ellerbrock

CONTRIBUTORS

4th Edition (2010)

Editors:

Becky Wong, M.D.
Kate Ellerbrock, M.D.
Aileen Adriano, M.D.

Resident Mentors:

Sarah Bain, M.D.
Christie Brown, M.D.
Dora Castaneda, M.D.
Michael Charles, M.D.
Kate Ellerbrock, M.D.
Erin Hennessey, M.D.
Jody Leng, M.D.
Javier Lorenzo, M.D.
David Medina, M.D.
Brett Miller, M.D.
John Peterson, M.D.
Rohith Piyaratna, M.D.
Becky Wong, M.D.
Andrew Wall, M.D.
Romy Yun, M.D.

2010 Faculty Mentors

Tim Angelotti, M.D., Ph.D.
Martin Angst, M.D., Ph.D.
Lindsay Atkinson, M.D.
Alex Butwick, M.D.
Divya Chander, M.D., Ph.D.
Larry Chu, M.D., M.S.
Jeremy Collins, M.D.
Ana Crawford, M.D.
Ali Djalali, M.D.
Anthony Doufas, M.D., Ph.D.
Ruth Fanning, M.D.
Sara Goldhaber-Fiebert, M.D.
Lee Hanowell, M.D.
Gill Hilton, M.D.
Jerry Ingrande, M.D., M.S.
Richard Jaffe, M.D., Ph.D.
Vivek Kulkarni, M.D., Ph.D.
Steve Lipman, M.D.
Alex Macario, M.D., MBA
Kevin Malott, M.D.
Diana McGregor, M.D.
John Nguyen, M.D.
Periklis Panousis, M.D.
Suma Ramzan, M.D.

Ed Riley, M.D.
Vanila Singh, M.D.
Pedro Tanaka, M.D., Ph.D.
Ying Ting, M.D.
Kimberly Valenta, M.D.
Karl Zheng, M.D.

3rd Edition (2009)

Editors:

Jessica Kentish, M.D.
William Hightower, M.D.
Tara Cornaby, M.D.

Resident Mentors and Contributing Authors:

Sarah Bain, M.D.
Marisa Brandt, M.D.
Erin Hennessey, M.D.
Billy Hightower, M.D.
Jesse Hill, M.D.
Meredith Kan, M.D.
Zoe Kaufenberg, M.D.
Jessica Kentish, M.D.
Zeest Khan, M.D.
Milo Lochbaum, M.D.
Nate Ponstein, M.D.
Tzevan Poon, M.D.

2009 Faculty Mentors

Aileen Adriano, M.D.
Tim Angelotti, M.D., Ph.D.
Jeremy Collins, M.D.
Tara Conaby, M.D.
Anthony Doufas, M.D., Ph.D.
Ruth Fanning, M.D.
Sara Goldhaber-Fiebert, M.D.
Cosmin Guta, M.D.
Leland Hanowell, M.D.
Vivek Kulkarni, M.D.
Hendrikus Lemmens, M.D.,
PhD.
Diana McGregor, M.D.
Alex Macario, M.D., MBA
Kevin Malott, M.D.
Ed Riley, M.D.

Cliff Schmiesing, M.D.
Pedro Tanaka, M.D.
Alex Tzabazis, M.D.

2nd Edition (2008)

Editors:

Jerry Ingrande, M.D.
Aileen Adriano, M.D.

Resident Mentors and Contributing Authors:

Rich Cano, M.D.
Jennifer Hah, M.D.
Alyssa Hamman, M.D.
Jenna Hansen, M.D.
Jerry Ingrande, M.D.
Zoe Kaufenberg, M.D.
Nate Kelly, M.D.
Eddie Kim, M.D.
Allegra Lobell, M.D.
Julianne Mendoza, M.D.
John Nguyen, M.D.
Katie Polhemus, M.D.
Nicolette Roemer, M.D.
Karl Zheng, M.D.

1st Edition (2007)

Editors:

Ryan Green, M.D., Ph.D.
Aileen Adriano, M.D.

Resident Mentors and Contributing Authors:

Lindsey Atkinson, M.D.
Melissa Ennen, M.D.
Ryan Green, M.D., Ph.D.
Jung Hong, M.D.
Jerry Ingrande, M.D.
Sam Mireles, M.D.
Vicki Ting, M.D.
Glenn Valenzuela, M.D.
Jerrin West, M.D.

KEY POINTS AND EXPECTATIONS

Key Points:

- The program will last 4 weeks.
- Mentors will consist of faculty members and senior residents (CA-2s and CA-3s).
- CA-1s scheduled to start in the Stanford GOR will be assigned a different mentor each week (CA-1s scheduled to begin at the Palo Alto VAMC or Santa Clara Valley Medical Center will be mentored according to local program goals and objectives).
- Faculty will provide one-on-one mentoring while senior residents will provide one-on-one mentoring with oversight by a supervising faculty member.
- Mentors (both faculty and residents) and CA-1s will take weekday call together. CA-1s will take call with their mentor, but only in a shadowing capacity; both mentor and CA-1 take DAC (day-off after call) together.
- All CA-1s (including those starting at Stanford, VAMC, and SCVMC) will receive the syllabus of intra-operative mini-lecture topics to be covered with their mentors. These mini-lectures provide goal-directed intra-operative teaching during the first month. CA-1s will document the completion of each mini-lecture by obtaining their mentors' initials on the "Checklist for CA-1 Mentorship Intra-operative Didactics."
- CA-1s will receive verbal feedback from their mentors throughout the week, as appropriate, and at the end of each week. Mentors will communicate from week to week to improve longitudinal growth and mentorship of the CA-1.

Expectations of CA-1 Residents:

- Attend the afternoon CA-1 Introduction to Anesthesia Lecture Series.
- Participate in goal-directed learning by completing the CA-1 Mentorship Intra-operative Didactics with your mentors.
- Discuss cases with your mentor the night before.
- Take weekday call with your mentor. You will be expected to stay as long as the ongoing cases are of high learning value. You will take DAC day off with your mentor.
- CA-1s at SUH are not expected to take weekend call with your mentor (for those at the Valley and VA, discuss with your mentor).

Expectations of Senior Resident Mentors:

- Senior mentors will take primary responsibility for discussing the case, formulating a plan, and carrying out the anesthetic with their CA-1; if concerns arise, the senior mentor will discuss the case with the covering faculty member.
- Instruct CA-1s in the hands-on technical aspects of delivering an anesthetic.
- Participate in goal-directed learning by completing the CA-1 Mentorship Intra-operative Didactics with your CA-1.
- Take weekday call with your CA-1. When you go home, your CA-1 goes home. When you have a DAC, your CA-1 has a DAC.
- Provide timely feedback to your CA-1 every day and at the end of the week.
- Provide continuity of teaching by communicating with the CA-1's other mentors.

Expectations of Faculty Mentors:

- Participate in goal-directed learning by completing the CA-1 Mentorship Intra-operative Didactics with your CA-1.
- Take weekday call with your CA-1. When you go home, your CA-1 goes home. When you have a DAC, your CA-1 has a DAC.
- Provide timely feedback to your CA-1 every day and at the end of the week.
- Provide continuity of teaching by communicating with the CA-1's other mentors.

GOALS OF THE CA-1 TUTORIAL MONTH

Anesthesia is a “hands-on” specialty. Acquiring the fundamental knowledge, as well as cognitive and technical skills necessary to provide safe anesthesia, are essential early on in your training. The CA-1 Mentorship Program and the CA-1 Introduction to Anesthesia Lecture Series will provide you with the opportunity to achieve these goals. The following are essential cognitive and technical skills that each CA-1 resident should acquire by the end of their first month.

I. Preoperative Preparation:

- a. Perform a complete safety check of the anesthesia machine.
- b. Understand the basics of the anesthesia machine including the gas delivery systems, vaporizers, and CO₂ absorbers.
- c. Set up appropriate equipment and medications necessary for administration of anesthesia.
- d. Conduct a focused history with emphasis on co-existing diseases that are of importance to anesthesia.
- e. Perform a physical examination with special attention to the airway and cardiopulmonary systems.
- f. Understand the proper use of laboratory testing and how abnormalities could impact overall anesthetic management.
- g. Discuss appropriate anesthetic plan with patient and obtain an informed consent.
- h. Write a pre-operative History & Physical with Assessment & Plan in the chart.

II. Anesthetic Management

- a. Placement of intravenous cannulae. Central venous catheter and arterial catheter placement are optional.
- b. Understanding and proper use of appropriate monitoring systems (BP, EKG, capnography, temperature, and pulse oximeter).
- c. Demonstrate the knowledge and proper use of the following medications:
 - i. Pre-medication: Midazolam
 - ii. Induction agents: Propofol, Thiopental, Etomidate
 - iii. Neuromuscular blocking agents: Succinylcholine and at least one non-depolarizing agent
 - iv. Anticholinesterase and Anticholinergic reversal agents: Neostigmine and Glycopyrrolate
 - v. Local anesthetics: Lidocaine
 - vi. Opioids: Fentanyl and at least one other opioid
 - vii. Inhalational anesthetics: Nitrous oxide and one other volatile anesthetic
 - viii. Vasoactive agents: Ephedrine and Phenylephrine
- d. Position the patient properly on the operating table.
- e. Perform successful mask ventilation, endotracheal intubation, and LMA placement.
- f. Recognize and manage cardiopulmonary instability.
- g. Spinal and epidural anesthesia are optional.
- h. Record intra-operative note and anesthetic data accurately, punctually, and honestly.

III. Post-operative Evaluation

- a. Transport a stable patient to the Post Anesthesia Care Unit (PACU)
- b. Provide a succinct anesthesia report to the PACU resident and nurse.
- c. Complete the anesthesia record with proper note.
- d. Leave the patient in a stable condition.
- e. Make a prompt post-operative visit and leave a note in the chart (optional but strongly encouraged).

CHECKLIST FOR CA-1 MENTORSHIP INTRAOPERATIVE DIDACTICS

(half of the CA1's will have ACRM on July 2, and the other half on July 6)

Mentors *initial* completed lectures

First Day July 1	_____	Discuss GOR Goals and Objectives for CA-1
	_____	Discuss etiquette in the OR
	_____	Discuss proper documentation
	_____	Discuss proper sign out
	_____	Discuss post-op orders
	_____	Machine check
Week One July 7-9	_____	Standard Monitors
	_____	Inhalational Agents
	_____	MAC & Awareness
	_____	IV Induction Agents
	_____	Rational Opioid Use
Week Two July 12-16	_____	Intra-operative Hypotension & Hypertension
	_____	Neuromuscular Blocking Agents
	_____	Difficult Airway Algorithm
	_____	Fluid Management
	_____	Transfusion Therapy
	_____	Hypoxemia
Week Three July 19-23	_____	Electrolyte Abnormalities
	_____	Hypothermia & Shivering
	_____	PONV
	_____	Extubation Criteria & Delayed Emergence
	_____	Laryngospasm & Aspiration
Week Four July 26-30	_____	Oxygen Failure in the OR
	_____	Anaphylaxis
	_____	ACLS
	_____	Malignant Hyperthermia
	_____	Perioperative Antibiotics

Standard Monitors

Basic Anesthetic Monitoring

ASA Standards for Basic Anesthetic Monitoring

STANDARD I

"Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics, and monitored anesthesia care."

STANDARD II

"During all anesthetics, the patient's **oxygenation, ventilation, circulation, and temperature** shall be continually evaluated."

OXYGENATION

- F_iO_2 Analyzer
- Pulse Oximetry

VENTILATION

- Capnography
- Disconnect alarm

CIRCULATION

- EKG
- Blood Pressure
- Pulse Oximetry

TEMPERATURE

- Temperature Probe

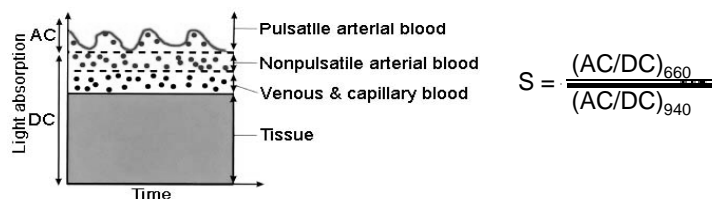
Pulse Oximetry

Terminology

- S_aO_2 (Fractional Oximetry) = $O_2Hb / (O_2Hb + Hb + MetHb + COHb)$
- S_pO_2 (Functional Oximetry/Pulse Oximetry) = $O_2Hb / (O_2Hb + Hb)$

Fundamentals

- The probe emits light at 660 nm (red, for Hb) and 940 nm (infrared, for O_2Hb); sensors detect the light absorbed at each wavelength.
- Photoplethysmography is used to identify arterial flow (alternating current = AC) and cancels out the absorption during non-pulsatile flow (direct current = DC); the patient is their own control!
- The S value is used to derive the S_pO_2 (S = 1:1 ratio = S_pO_2 85%).



Pulse Oximetry

Pearls

- Methemoglobin (MetHb) - Similar light absorption at 660 nm and 940 nm (1:1 ratio); at high levels, S_pO_2 approaches 85%.
- Carboxyhemoglobin (COHb) - Similar absorbance to O_2Hb . At 50% COHb, S_aO_2 = 50% on ABG, but S_pO_2 may be 95%, thus producing a falsely HIGH S_pO_2 .
- Other factors producing a falsely LOW S_pO_2 = dyes (methylene blue > indocyanine green > indigo carmine), blue nail polish, shivering, ambient light.
- Factors with NO EFFECT on S_pO_2 = bilirubin, HbF, HbS, SuHb, acrylic nails, fluorescein dye.
- Cyanosis - clinically apparent with 3 g/dl desaturated Hb. At Hb = 15 g/dl, cyanosis occurs at S_aO_2 = 80%; at Hb = 9 g/dl (i.e. anemia), cyanosis occurs at S_aO_2 = 66%.

EKG

3-Electrode System

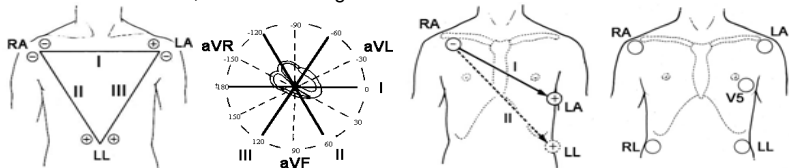
- Allows monitoring of Leads I, II, and III, but only one lead (i.e. electrode pair) can be examined at a time while the 3rd electrode serves as ground.
- Lead II is best for detecting P waves and sinus rhythm.

Modified 3-Electrode System

- If you have concerns for anterior wall ischemia, move L arm lead to V5 position, and monitor Lead I for ischemia.

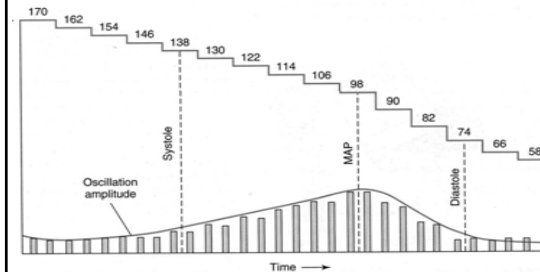
5-Electrode System

- Four limb leads + V5 (left anterior axillary line, 5th ICS), allows monitoring of 7 leads simultaneously.
- V5 is 75% sensitive for detecting ischemic events; II + V5 is 80% sensitive; II + V4 + V5 together is 98% sensitive.



Noninvasive Blood Pressure

- Automated, microprocessor-assisted interpretation of oscillations in the NIBP cuff.
- MAP is primary measurement; SBP and DBP are derived from algorithms.
- Bladder should encircle ≥50% of extremity; width should be 20-50% greater than diameter of extremity.
- Cuff too small = falsely HIGH BP. Cuff too big = falsely LOW BP.



FYI:

$$\text{MAP} = \frac{\text{SBP} + 2\text{DBP}}{3}$$

Arterial Blood Pressure

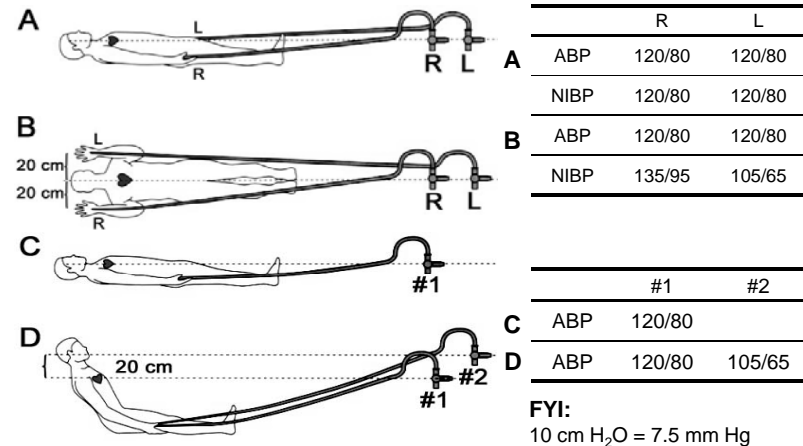
Indications

- Moment-to-moment BP changes anticipated and rapid detection is vital.
- Planned pharmacologic or mechanical manipulation.
- Repeated blood sampling.
- Failure of NIBP.
- Supplementary diagnostic information (e.g. perfusion of dysrhythmic activity, volume status, IABP).

Transducer Setup

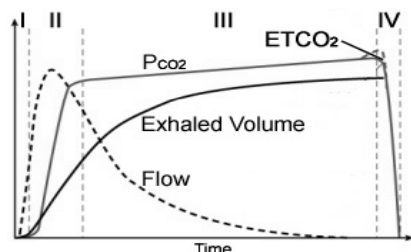
- Zeroing = exposes the transducer to air-fluid interface at any stopcock, thus establishing P_{atm} as the "zero" reference pressure.
- Leveling = assigns the zero reference point to a specific point on the patient; by convention, the transducer is "leveled" at the right atrium.

Effect of Patient & Transducer Position on BP Measurement



Capnography

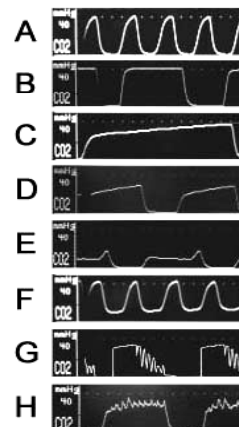
- Measures exhaled CO_2 (and other gases).
- Time delay exists due to length and volume of sample tube as well as sampling rate (50-500 ml/min).



Capnogram Phases

- Dead space gas exhaled
- Transition between airway and alveolar gas
- Alveolar plateau
- Inspiration

Capnography



Example Traces

- Spontaneous ventilation
- Mechanical ventilation
- Prolonged exhalation (spontaneous)
- Emphysema
- Sample line leak
- Exhausted CO_2 absorbant
- Cardiogenic oscillations
- Electrical noise

Temperature

Monitoring is required “when clinically significant changes in body temperature are intended, anticipated, or suspected.”

Sites

- Pulmonary artery = “Core” temperature (gold standard)
- Tympanic membrane - correlates well with core; approximates brain/hypothalamic temperature
- Esophagus - correlates well with core
- Nasopharyngeal - correlates well with core and brain temperature
- Rectal - not accurate (temp affected by LE venous return, enteric organisms, and stool insulation)
- Bladder - approximates core when urine flow is high
- Axillary - inaccurate; varies by skin perfusion
- Skin - inaccurate; varies by site
- Oropharynx – good estimate of core temperature; recent studies show correlation with tympanic and esophageal temperatures

References

- ASA. Standards for basic anesthetic monitoring (http://www.asahq.org/publications_and_services/standards/02.pdf). 2005.
- Mark JB, and Slaughter TF. Cardiovascular monitoring. In Miller RD (ed), *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- Moon RE, and Camporesi EM. Respiratory monitoring. In Miller RD (ed), *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill Companies, Inc., 2006.
- Narang J, and Thys D. Electrocardiographic monitoring. In Ehrenwerth J, and Eisenkraft JB (eds), *Anesthesia Equipment: Principles and Applications*. St. Louis: Mosby, 1993.
- Skeehan TM and Jopling M. Monitoring the cardiac surgical patient. In Hensley FA, Martin DE, and Gravlee GP (eds), *A Practical Approach to Cardiac Anesthesia*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.

Inhalational Agents

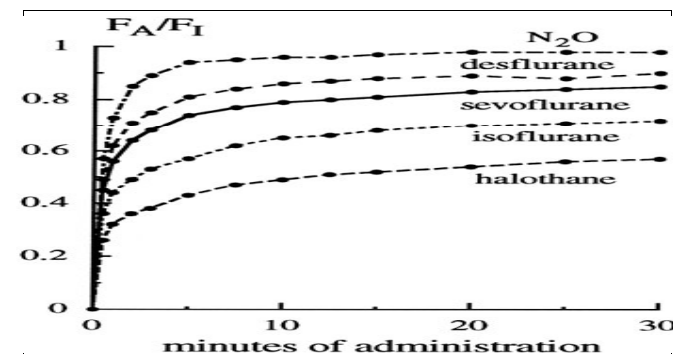
Pharmacokinetics

- The pharmacokinetics of inhalational agents is divided into four phases
 - Absorption
 - Distribution (to the CNS)
 - Metabolism (minimal)
 - Excretion (minimal)
- The ultimate goal is to establish a particular partial pressure of an agent in the lungs
 - This partial pressure will equilibrate with the CNS tissue to produce an anesthetized state
- At equilibrium the following applies

$$P_{\text{CNS}} = P_{\text{blood}} = P_{\text{Alveoli}}$$

Uptake and Distribution

- Inhalational anesthetic uptake is commonly followed by the ratio of fractional concentration of alveolar anesthetic to inspired anesthetic (F_A/F_I)
- Uptake into the bloodstream is the primary determinant of F_A
- The greater the uptake (in blood), the slower the rate of rise of F_A/F_I
 - Uptake is proportional to tissue solubility
 - The gases with the lowest solubilities in blood (i.e. desflurane) will have the fastest rise in F_A/F_I
 - They also have the fastest elimination
 - Rate of rise of F_A/F_I is proportional to clinical effect (i.e. the faster the rate of rise, the faster the induction and also elimination)



The rise in alveolar (F_A) anesthetic concentration toward the inspired (F_I) concentration is most rapid with the least soluble anesthetics, nitrous oxide, desflurane, and sevoflurane. It rises most slowly with the more soluble anesthetics, for example, halothane. All data are from human studies. (Adapted from Yasuda N, Lockhart SH, Eger EI II *et al*: Comparison of kinetics of sevoflurane and isoflurane in humans. *Anesth Analg* 72:316, 1991; and Yasuda N, Lockhart SH, Eger EI II *et al*: Kinetics of desflurane, isoflurane, and halothane in humans. *Anesthesiology* 74:489, 1991.)

Factors That Increase or Decrease the Rate of Rise of F_A/F_I

•INCREASE •DECREASE		
Low λ_B	High λ_B	The lower the blood:gas solubility, the faster the rise in F_A/F_I
Low Q	High Q	The lower the cardiac output, the faster the rise in F_A/F_I
High V_A	Low V_A	The higher the minute ventilation, the faster the rise in F_A/F_I
High $(P_A - P_v)$	Low $(P_A - P_v)$	At the beginning of induction, P_v is zero but rises rapidly (thus $[P_A - P_v]$ falls rapidly) and F_A/F_I increases rapidly. Later, during induction and maintenance, P_v rises more slowly so F_A/F_I rises more slowly.

Parameters as described in Equation 15-16: λ_B , blood solubility; Q , cardiac output; V_A , minute ventilation; P_A , P_v , pulmonary arterial and venous blood partial pressure. (Clinical Anesthesia 5th Edition; Barash, P.; Lippincott Williams and Wilkins; 2006)

Pharmacodynamics

- All inhalational agents decrease CMO_2 and increase CBF (via direct vasodilation)
 - Increases in CBF can in turn increase ICP
- All agents cause a dose-related decrease in blood pressure
- All agents produce muscle relaxation
- The older inhalational agents (halothane, enflurane) cause decreases in myocardial contractility
 - The newer agents have little to no effect
- All inhalational agents produce a dose-dependent depression of the ventilatory response to hypercarbia and hypoxia

Nitrous Oxide

- Low potency (MAC 104%)
- Insoluble in blood
 - Facilitates rapid uptake and elimination
- Commonly administered as an anesthetic adjuvant
- Does not produce skeletal muscle relaxation
- Can potentially contribute to PONV
- Can diffuse into air filled cavities and cause expansion of air filled structures (pneumothorax, bowel, middle ear, ET tube balloons, etc.)
 - Often contraindicated in these settings

Isoflurane

- Highly pungent
- Second most potent of the clinically used inhalational agents (MAC 1.2%)
- Preserves flow-metabolism coupling in the brain
 - Highly popular for neuroanesthesia
- Has been implicated for causing "coronary steal"
 - Dilation of "normal" coronary arteries causing blood to be diverted away from maximally dilated, stenotic vessels to vessels with more adequate perfusion
- Causes vasodilation
 - Decreases BP
 - Increases CBF (usually seen at 1.6 MAC)
 - Minimal compared to halothane
 - Increases ICP (usually at above 1 MAC; short lived)
 - Minimal compared to halothane

Sevoflurane

- Half as potent as isoflurane (MAC 1.8%)
- Quick uptake and elimination
- Sweet smelling, non-pungent
- Quick uptake and sweet smell make this agent very popular for inhalational induction
- Potent bronchodilator
- Can form CO in dessicated CO₂ absorbent
 - Can cause fires
- Forms Compound A in CO₂ absorbent
 - Recommended to keep fresh gas flows >2 L/min

Desflurane

- Blood:gas solubility coefficient equal to N₂O
- Very quick uptake and elimination
- Low potency (MAC 6.6%)
- High vapor pressure
 - Must be stored in a heated, pressurized vaporizer
- Very pungent
 - Can cause breath-holding, bronchospasm, laryngospasm when administered to an awake patient via face mask
- Can form CO in dessicated CO₂ absorbent
- Can cause an increased sympathetic response (tachycardia, hypertension) when inspired concentration is increased rapidly

References

1. Clinical Anesthesia 5th Edition; Barash P., Cullen B., Stoelting R.; Lippincott Williams and Wilkins, 2006
2. Miller's Anesthesia 6th edition; Miller R.; Churchill Livingstone, 2005
3. The Pharmacology of Inhalational Anesthetics 3rd edition; Eger E., Eisenkraft J., Weiskopf R.; Library of Congress, 2003
4. Yasuda N, Lockhart SH, Eger E *et al*: Comparison of kinetics of sevoflurane and isoflurane in humans. Anesth Analg 72:316, 1991
5. Yasuda N, Lockhart SH, Eger E *et al*: Kinetics of desflurane, isoflurane, and halothane in humans. Anesthesiology 74:489, 1991

It was the first case in the morning. I checked the gases and they were all filled up to the top. 10 minutes into the case, half the sevo was gone and I was running low flows. I was like what the heck! My med student starts coughing, I had a big headache, the surgeons didn't say a word, which was weird because that surgeon usually says a lot. The med student also had asthma and said something was making her cough. I checked for a leak in my circuit, checked my numbers, everything was fine. I called for an anesthesia tech and they checked the caps. Turns out that the anesthesia tech the day before hadn't screwed the cap back on tightly where you refill the stuff. The room was gassed.

MAC & Awareness

Minimum Alveolar Concentration

Alveolar concentration of a gas at which 50% of subjects do not respond to surgical incision

Important Points

- Remarkably consistent across species.
- MAC is a population average; not a true predictor of an individual's response.
- MAC is an ED_{50} concentration. The ED_{95} is $\pm 25\%$, so at 1.3 MAC, 95% of patients will not respond to incision.
- MAC values are additive (e.g. 0.5 MAC isoflurane + 0.5 MAC N_2O = 1 MAC)

MAC of Inhaled Anesthetics

Gas	Blood:Gas Partition Coefficient	MAC*
Halothane	2.4	0.75%
Enflurane	1.9	1.7%
Isoflurane	1.4	1.2%
Sevoflurane	0.65	2.0%
N_2O	0.47	104%
Desflurane	0.42	6.0%

*MAC values for adults 36-49 years old

- MAC is an indicator of gas potency.
- The blood:gas partition coefficient is an indicator of solubility, which affects the rate of induction and emergence; it is NOT related to MAC.

More MAC Definitions

MAC-Awake (a.k.a. MAC-Aware)

- The MAC necessary to prevent response to verbal/tactile stimulation.
- Volatiles: ~ 0.4 MAC; N_2O : ~ 0.6 MAC

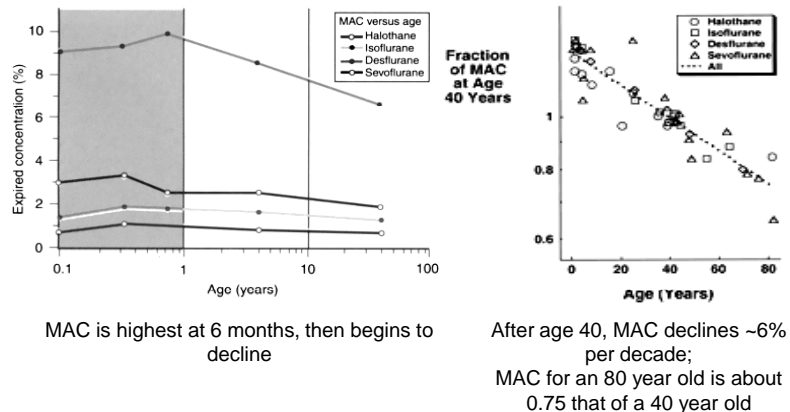
MAC-BAR

- The MAC necessary to “blunt the autonomic response” to a noxious stimulus
- ~ 1.6 MAC

MAC-EI

- The MAC necessary to prevent laryngeal response to “endotracheal intubation”
- ~ 1.3 MAC

Effect of Age on MAC



Factors Increasing MAC

- Drugs increasing central catecholamines:
 - MAOIs, TCAs
 - Acute cocaine and amphetamine use
 - Ephedrine
 - Levodopa
- Hyperthermia
- Hypernatremia
- Chronic EtOH abuse
- Genetic factors
 - Redheaded females have a 19% increased MAC requirement compared to brunettes.

Factors Decreasing MAC

- Drugs decreasing central catecholamines:
 - Reserpine, α -methyldopa
 - Chronic amphetamine abuse
- Other drugs:
 - Opioids, benzodiazepines, barbiturates, α_2 -agonists (clonidine, dexmedetomidine), ketamine, lidocaine, lithium, verapamil, hydroxyzine.
- Acute EtOH intoxication
- Pregnancy (after 8-12 weeks gestation)
- Hypothermia (\downarrow 50% per 10°C)
- Hypotension (MAP < 40 in adult)
- Hypoxemia ($P_{aO_2} < 38$ mm Hg) or hypercarbia ($P_{aCO_2} > 95$ mm Hg)
- Hyponatremia
- Metabolic acidosis
- Anemia (Hct < 10%)

Awareness

- Very rare
- Most common sensation is hearing voices
- Mostly occurs during induction or emergence
- More common in high-risk surgeries where deep anesthesia may be dangerous to an unstable patient (e.g. trauma, cardiac, cesarean section)
- Early counseling after an episode is very important
- Patient handout available at:
www.asahq.org/patientEducation/Awarenessbrochure.pdf

Signs of Light Anesthesia

- Increase in HR or BP by 20% above baseline
- Tearing
- Dilated pupils
- Coughing or bucking
- Patient movement
- Signs of consciousness on EEG monitor (Bispectral Index or Patient State Index)

BIS & PSI

- Both use EEG monitoring and algorithms to produce numbers (0-100) relating to depth of anesthesia.
 - 65-85 = sedation
 - 40-65 = general anesthesia
 - <40 = too deep
- Both have been shown to be fairly good predictors of loss and regaining consciousness
- Interpatient variability exists
- Both have a noticeable time lag
- BIS is affected by electrocautery more than PSI

Management

If you suspect your patient may be aware:

- Immediately deepen the anesthetic with fast-acting agents (e.g. propofol).
- Talk to the patient, reassure them that everything is OK (hearing is the last sense to be lost).
- Consider a benzodiazepine for amnesia.
- Talk to the patient after the case to assess if they had any awareness.
- Set up counseling if necessary.
- Contact Risk Management (potential lawsuit?)

References

- ASA and AANA. *Patient awareness under general anesthesia - what is it?* (www.asahq.org/patientEducation/Awarenessbrochure.pdf), 2005.
- Chen X, Tang J, et al. 2002. A comparison of PSI and BIS values during the perioperative period. *Anesth Analg*, **95**: 1669-74.
- Ebert TJ. Inhalation anesthesia. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.
- Evers AS and Crowder CM. Cellular and molecular mechanisms of anesthesia. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill Companies, Inc., 2006.

IV Induction Agents

Mechanism of Action

- It is widely believed that IV anesthetics exert their sedative and hypnotic effects via their interaction with GABA
 - GABA is the primary inhibitory neurotransmitter in the CNS
 - Activation of receptor causes increased Chloride conductance and therefore, hyperpolarization
- Propofol and the barbiturates decrease the rate of dissociation of GABA and its receptor
- Benzodiazepines increases the efficiency of GABA-receptor coupling

Pharmacokinetic Values for the Currently Available Intravenous Sedative-Hypnotic Drugs

•DRUG NAME	•DISTRIBUTION HALF-LIFE (min)	•PROTEIN BINDING (%)	•DISTRIBUTION VOLUME AT STEADY STATE (L/kg)	•CLEARANCE (mL/kg/min)	•ELIMINATION HALF-LIFE (h)
Thiopental	2-4	85	2.5	3.4	11
Methohexital	5-6	85	2.2	11	4
Propofol	2-4	98	2-10	20-30	4-23
Midazolam	7-15	94	1.1-1.7	6.4-11	1.7-2.6
Diazepam	10-15	98	0.7-1.7	0.2-0.5	20-50
Lorazepam	3-10	98	0.8-1.3	0.8-1.8	11-22
Etomidate	2-4	75	2.5-4.5	18-25	2.9-5.3
Ketamine	11-16	12	2.5-3.5	12-17	2-4

(Clinical Anesthesia 5th Edition; Barash, P.; Lippincott Williams and Wilkins; 2006)

Induction Characteristics and Dosage Requirements for the Currently Available Sedative-Hypnotic Drugs

•DRUG NAME	•INDUCTION DOSE (mg/kg)	•ONSET (sec)	•DURATION (min)	•EXCITATORY ACTIVITY [*]	•PAIN ON INJECTION [*]	•HEART RATE [†]	•BLOOD PRESSURE [†]
Thiopental	3-6	<30	5-10	+	0-+	↑	↓
Methohexital	1-3	<30	5-10	++	+	↑↑	↓
Propofol	1.5-2.5	15-45	5-10	+	++	0-↓	↓↓
Midazolam	0.2-0.4	30-90	10-30	0	0	0	0/↓
Diazepam	0.3-0.6	45-90	15-30	0	+/+++	0	0/↓
Lorazepam	0.03-0.06	60-120	60-120	0	++	0	0/↓
Etomidate	0.2-0.3	15-45	3-12	+++	+++	0	0
Ketamine	1-2	45-60	10-20	+	0	↑↑	↑↑

^{*}0 = none; + = minimal; ++ = moderate; +++ = severe.

[†]↓ = decrease; ↑ = increase.

(Clinical Anesthesia 5th Edition; Barash, P.; Lippincott Williams and Wilkins; 2006)

Pharmacodynamics

- The principle pharmacologic effect of IV anesthetics is to produce increasing sedation and eventually hypnosis
- All hypnotics also effect other major organ systems
 - They produce a dose-dependant respiratory depression (exception: Ketamine)
 - They produce hypotension and cardiac depression (Etomidate causes the least cardiac depression)
- Profound hemodynamic effects can be seen with hypovolemia as a higher drug concentration is achieved at the central compartment
 - A large hemodynamic depressant effect can be seen in the elderly and those with pre-existing cardiovascular disease
 - These patients often require a decreased dose requirement

Drug	Dose (mg/kg)	Effects	Pearls
Propofol	1.5-2.5	<u>Neuro:</u> Decreases cerebral metabolic O ₂ requirements, cerebral blood flow, intracranial pressure <u>CV:</u> Decreases SVR, direct myocardial depressant <u>Pulm:</u> Dose dependant respiratory depression (apnea in 25-35% of patients)	-Pain on injection(32-67%) -can be attenuated with lidocaine -antiemetic properties -anticonvulsant properties
Etomidate	0.2-0.3	<u>Neuro:</u> Decreases CMRO ₂ , CBF, ICP <u>CV:</u> Maintains hemodynamic stability (minimal cardiac depression) <u>Pulm:</u> minimal respiratory depression (no histamine release)	-Pain on injection -High incidence of PONV -myoclonus -inhibits adrenocortical axis
Thiopental	3-5	<u>Neuro:</u> Decreases CMRO ₂ , CBF, ICP <u>CV:</u> Decreases SVR, direct myocardial depressant <u>Pulm:</u> Dose dependant respiratory depression	-anticonvulsant properties -can precipitate when injected with acidic fluids (i.e LR)
Ketamine	1-2	<u>Neuro:</u> Increases CMRO ₂ , CBF, ICP <u>CV:</u> Cardio-stimulating effects (negatively effects myocardial supply-demand) <u>Pulm:</u> minimal respiratory depression; bronchodilation; most likely of all to protect airway reflexes	-good analgesic effects -intrinsic myocardial depressant effects which may be unmasked with depleted catecholamines

Propofol

- Produced in an egg lecithin emulsion because of its high lipid solubility
- Pain on injection occurs in 33-67% of subjects
- Can be attenuated with lidocaine or administering the drug in a larger vein
- Induction dose 1.5-2.5 mg/kg
 - Children require higher doses (larger Volume of Distribution)
 - Elderly require lower doses (smaller Volume of Distribution)
- Decreases cerebral metabolic O₂ requirements, cerebral blood flow, intracranial pressure
- Decreases SVR, direct myocardial depressant
- Dose-dependent respiratory depression
- Has anti-emetic properties
- Anticonvulsant properties

Etomidate

- Can produce pain on injection
- Induction dose 0.2-0.3 mg/kg
- Myoclonus common upon injection
- Decreases cerebral metabolic O₂ requirements, cerebral blood flow, intracranial pressure
- Maintains hemodynamic stability (even in the presence of pre-existing disease)
 - Does not induce histamine release
- Inhibits adrenocortical synthetic function
 - Inhibition for 5-8 hours even after a single induction dose!
- High incidence of PONV

Thiopental

- Highly alkaline (pH 9)
- Can precipitate in acidic solutions
 - DO NOT MIX with Rocuronium
- Induction dose 3-5 mg/kg
- Rapidly redistributed into peripheral compartments
- Accounts for its short duration of action
- Larger doses can saturate the peripheral compartments resulting in a prolonged duration of action
- Decreases CMRO₂, CBF, ICP
 - Causes EEG burst suppression in larger doses (often used for neurosurgical procedures)
- Decreases SVR, direct myocardial depressant
- Anticonvulsant activity
 - Exception: Methohexital

Ketamine

- Produces a dissociative anesthetic state
 - Profound analgesia and amnesia despite maintenance of consciousness
 - High incidence of psychomimetic reactions
- Induction dose 1-2 mg/kg
- NMDA antagonist
- Increases CMRO₂, CBF, ICP
 - Contraindicated in neurosurgical procedures
- Most likely to preserve airway reflexes among the IV anesthetics
- Minimal respiratory depression
- Cardio-stimulating effects
 - Can be unmasked in patients with increased sympathetic outflow
 - Negatively effects myocardial oxygen supply-demand ratio
- Causes bronchodilation

Midazolam

- All benzodiazepines have anxiolytic, amnestic, sedative, hypnotic, anticonvulsant properties
- Premedication dose 0.04-0.08 mg/kg
- Decreases CMRO₂, CBF, ICP
 - Does not produce EEG burst suppression
- Causes dose-dependant respiratory depression
 - Exaggerated when combined with opioids
- Flumazenil is a specific antagonist
 - Very short acting
 - 45-90 minutes of action following 1-3 mg dose
 - May see re sedation as benzodiazepine is eliminated more slowly compared to effects of flumazenil

References

1. Clinical Anesthesia 5th Edition; Barash P., Cullen B., Stoelting R.; Lippincott Williams and Wilkins, 2006
2. Miller's Anesthesia 6th edition; Miller R.; Churchill Livingstone, 2005

Rational Opioid Use

Basic Opioid Pharmacology

- Analgesia produced by mu (μ) opioid receptor agonism in the brain (periaqueductal gray matter) and spinal cord (substantia gelatinosa).
- Well-known side effect profile:
 - Sedation, respiratory depression
 - Itching, nausea, ileus, urinary retention
 - Bradycardia, hypotension
 - Miosis, chest wall rigidity
- Opioids are hemodynamically stable when given alone, but cause \downarrow CO, SV, and BP in combination with other anesthetics.
- Reduces MAC of volatile anesthetics.

Opioids

Morphine

- Slow peak time (~80% effect at 15 minutes, but peak analgesic effect is at ~90 minutes).
- Active metabolite, morphine-6-glucuronide, has analgesic properties and is renally excreted (not clinically relevant unless patient has renal failure)
- Can cause histamine release.

Hydromorphone (Dilaudid)

- “A rapid onset morphine.” Peak effect in 5-10 minutes.
- About 8-fold more potent than morphine (i.e. 1 mg Dilaudid = 8 mg morphine)
- No active metabolites, no histamine release.
- Good choice for postop analgesia and PCA.

Opioids

Fentanyl

- Fast onset & short duration of action (peak effect at 3-5 minutes; effect site half-life ~30 minutes).
- ~100-fold more potent than morphine.
- Very cheap.

Sufentanil

- Fast onset, but slightly slower than fentanyl
- 10-fold more potent than fentanyl (i.e. 5 mcg sufentanil = 50 mcg fentanyl).
- More rapid recovery than fentanyl.

Opioids

Alfentanil

- Fastest onset time of all opioids (~90 seconds); pKa = 6.5, so it crosses the blood-brain barrier rapidly.
- Also causes more N/V, chest wall rigidity, and respiratory depression.
- Brief duration of action due to redistribution.

Remifentanil

- Peak effect time ~90 seconds
- Unique pharmacokinetics - metabolized by plasma esterases.
- Short context-sensitive half-time after termination of infusion with predictable offset in ~5-10 minutes.

Opioids

Meperidine (Demerol)

- Originally discovered as a local anesthetic ("pethidine")
- Peak effect in 15 minutes, lasts 2-4 hours.
- Active metabolite (normeperidine) lowers the seizure threshold; renally excreted.
- Useful for treating shivering.
- Anticholinergic side effects: tachycardia
- Avoid using with MAOIs; can cause CNS excitation (agitation, hyperpyrexia, rigidity) and/or CNS depression (hypotension, hypoventilation, coma)
- Causes histamine release.
- Has a euphoric effect with less respiratory depression than other opioids.

Comparison of Peak Effect Times



Rational Opioid Use

Note: All anesthesiologists (attendings & residents alike) have different theories and opinions on the optimal choice and dose of opioids in different situations. The strategies presented here are simply suggestions, something to get you thinking rationally about how and when you use opioids for analgesia. Discuss the merits of these strategies with your attending before or during each case, but do not take these suggestions as firm guidelines for how all anesthetics should be done!

With that disclaimer in mind, continue reading...

Strategies for Opioid Use

- For a standard GETA induction, use fentanyl to blunt the stimulation caused by DL and intubation.
- For brief, intense stimulation (e.g. retrobulbar block, Mayfield head pins, rigid bronchoscopy), consider a bolus of short-acting opioid like remifentanyl or alfentanil.
- For intraop analgesia:
 - Fentanyl is rapidly titratable, but requires frequent redosing; it may be more “forgiving” if overdosed.
 - Morphine has a long onset time to peak effect, but gives prolonged analgesia during the case and into the postop period.
 - Hydromorphone is rapidly titratable (like fentanyl) with prolonged analgesia (like morphine).

Strategies for Opioid Use

- For ENT cases, consider an opioid infusion (e.g. remifentanyl, alfentanil, sufentanil, or fentanyl):
 - Stable level of analgesia
 - Induced hypotension
 - “Narcotic wakeup” reduces bucking on ETT
 - Smooth transition to postop analgesia
- For chronic opioid users (e.g. methadone, MS Contin, OxyContin, etc.), continue the patient’s chronic opioid dose intraop PLUS expect higher opioid requirements for their acute pain.
- Use morphine and meperidine cautiously in renal patients (renal excretion of active metabolites)!

Strategies for Opioid Use

- Meperidine is usually reserved for treatment/prevention of postoperative shivering.
- For postop pain control (i.e. PACU):
 - Consider fentanyl (rapid onset, easily titratable, cheap, and the nurses are familiar with its use).
 - Consider hydromorphone (rapid onset, easily titratable, prolonged effect, nurses are familiar with its use, and it is a good transition to PCA).
 - If surgery is ambulatory and/or patient is tolerating POs, give Vicodin.

References

- Fukuda K. Intravenous opioid anesthetics. In Miller RD (ed), *Miller’s Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- Gustein HB and Akil H. Opioid analgesics. In Hardmann JG, Limbird LE, and Goodman Gilman A (eds), *Goodman & Gilman’s The Pharmacological Basis of Therapeutics*, 10th ed. New York: McGraw-Hill, 2001.
- Saidman L and Shafer S. 2005. “Rational Use of Opioids: Intraoperative and Postoperative,” presented at Stanford University Department of Anesthesia Grand Rounds, July 18, 2005.

Intraoperative Hypotension & Hypertension

Determinants of Blood Pressure

Blood Pressure (BP)

- BP represents the force exerted by circulating blood on the walls of blood vessels.
- A product of 1) cardiac output and 2) vascular tone.

Cardiac Output (CO)

- $CO = HR \times SV$

Heart Rate (HR)

- Dependent on the interplay between the sympathetic and parasympathetic nervous systems.
- In infants, SV is fixed, so CO is dependent on HR.
- In adults, SV plays a much more important role, particularly when increasing HR is not favorable.

Determinants of Blood Pressure

Stroke Volume (SV)

- Dependent on preload, afterload, and myocardial contractility.

Preload

- Volume of blood in the ventricle at end-diastole (LVEDV)

Afterload

- Resistance to ejection of blood from the ventricle
- SVR accounts for 95% of the impedance to ejection
- $SVR = [(MAP - CVP) \times 80] \div CO$

Contractility

- The force and velocity of ventricular contraction when preload and afterload are held *constant*.
- Ejection fraction (EF) is one of the most clinically useful indices of contractility (normal EF is ~60%).

Components of Blood Pressure

Systolic Blood Pressure (SBP)

- Highest arterial pressure in the cardiac cycle.
- Dicrotic notch = a small notch in the invasive arterial pressure curve that represents closure of the aortic valve, producing a brief period of retrograde flow.

Diastolic Blood Pressure (DBP)

- Lowest arterial pressure in the cardiac cycle

Mean Arterial Pressure (MAP)

- $MAP = 2/3 DBP + 1/3 SBP$, or $(2 \times DBP + SBP) \div 3$

Components of Blood Pressure

Pulse Pressure

- **PP = SBP - DBP**
- Normal PP is ~40 mm Hg at rest, and up to ~100 mm Hg with strenuous exercise.
- Narrow PP (e.g. < 25 mm Hg) = may represent aortic stenosis, coarctation of the aorta, tension pneumothorax, myocardial failure, shock, or damping of the system.
- Wide PP (e.g. > 40 mm Hg) = aortic regurgitation, atherosclerotic vessels, PDA, high output state (e.g. thyrotoxicosis, AVM, pregnancy, anxiety)

Blood Pressure Measurement

Non-Invasive Blood Pressure (NIBP)

- Oscillometric BP determination: oscillations in pressure are detected through the cuff as it deflates.
- MAP is measured as the largest oscillation; it is the most accurate number produced by NIBP.
- SBP and DBP are calculated by proprietary algorithms in the machine.

Invasive Arterial Blood Pressure (IABP)

- Most accurate method of measuring BP.
- If system is zeroed, leveled, and properly dampened, SBP, DBP, and MAP are very accurate.

Intraoperative Hypertension

- “Light” anesthesia
- Pain
- Chronic hypertension
- Illicit drug use (e.g. cocaine, amphetamines)
- Hypermetabolic state (e.g. MH, thyrotoxicosis, NMS)
- Elevated ICP (Cushing’s triad: HTN, bradycardia, irregular respirations)
- Autonomic hyperreflexia (spinal cord lesion > T5 = severe; < T10 = mild)
- Endocrine disorders (e.g. pheochromocytoma, hyperaldosteronism)
- Hypervolemia
- Drug contamination - intentional (e.g. local anesthetic + Epi) or unintentional (e.g. “Roc-inephine”)

Treatment of Hypertension

- Temporize with fast-onset, short-acting drugs, but ultimately diagnose and treat the underlying cause.
- Pharmacologic Interventions:
 - Volatile anesthetics (cause vasodilation while deepening anesthetic)
 - Opioids (treat pain and deepen the anesthetic)
 - Propofol (quickly sedates the “light” patient; also a vasodilator)
 - Beta-blockers (e.g. esmolol, labetalol, metoprolol)
 - Vasodilators (e.g. hydralazine, NTG, SNP)

Intraoperative Hypotension

- Excessive depth of anesthesia
 - Overdose of induction agent, volatile, or narcotic.
- Inadequate Preload (“the tank is empty”)
 - Hypovolemic shock (hypovolemia, anemia)
 - Increased intrathoracic pressure (e.g. excessive PEEP, I:E ratio, PTX, caval compression, chronic HTN)
- Reduced Afterload
 - Vasodilated states (e.g. liver failure, sepsis/SIRS/shock, anaphylaxis)
 - Depleted catecholamine states (e.g. adrenal suppression from chronic steroid use, methamphetamines, cocaine)
- Diminished Afterload
 - Acute MI, non-perfusing arrhythmia, cardiomyopathies, valvulopathies
 - Pulmonary HTN (decreases LVEDV)

Treatment of Hypotension

- Temporize with fast-onset, short-acting drugs, but ultimately diagnose and treat the underlying cause.
- Turn down the anesthetic (2 MAC? Too much!)
- Volume
 - Reevaluate EBL; replace with crystalloid, colloid, or blood, as needed.
 - Reevaluate patient’s fluid status (deficit, maintenance, and ongoing losses; urine output?).
 - Consider CVP, PAC, or TEE
- Ventilation
 - Reduce PEEP to improve venous return.
 - Decrease I:E ratio to shorten inspiratory time.
 - Rule out PTX
- Metabolic
 - Treat acidosis and/or hypocalcemia

Treatment of Hypotension

- Drugs
 - **Phenylephrine** (Neosynephrine) = α_1 agonist
 - Direct vasoconstrictor
 - Use in vasodilated state with tachycardia
 - Can cause reflex bradycardia
 - **Ephedrine** = α_1 , β_1 , and β_2 (less so) agonist
 - Direct and indirect adrenergic stimulation via NE release
 - Use in vasodilated, bradycardic, low CO states
 - **Epinephrine** = β_1 , α_1 , α_2 , and β_2 agonist
 - Endogenous catecholamine
 - Causes vasoconstriction and increased CO.
 - **Inotropes** (in low CO states)
 - Dopamine, Epinephrine, Milrinone, Dobutamine
 - **Stress-dose steroids** - consider 100 mg hydrocortisone if steroids taken in past 6 months.

References

- Costanzo LS. *Physiology*, 1st ed. Philadelphia: WB Saunders Company, 1998.
- Lawson NW and Johnson JO. Autonomic nervous system: physiology and pharmacology. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.
- Stoelting RK and Miller RD. *Basics of Anesthesia*, 4th ed. Philadelphia: Churchill Livingstone, 2000.

Neuromuscular Blocking Agents

Succinylcholine

- Structure = 2 adjoined ACh molecules!
- Mechanism of action is by ACh receptor activation and prolonged muscle depolarization.
- Dose: 1 to 1.5 mg/kg for intubation.
- Onset within 30-60 seconds and duration ~10 minutes depending on dose.
- Elimination by diffusion away from NMJ and metabolism by pseudocholinesterase (a.k.a. plasma cholinesterase)
 - Atypical pseudocholinesterase (genetic defect) can significantly prolong SCh block; enzyme activity measured by the “dibucaine number”:
 - Normal = 80 (i.e. dibucaine inhibits 80% of activity)
 - Heterozygote (1:480) = 50; block lasts ~30 minutes
 - Homozygote (1:3200) = 20; block lasts 6-8 hours

Succinylcholine: Adverse Effects

Hyperkalemia

- Can increase K⁺ by 0.5-1 mEq/L
- Long list of comorbid contraindications (e.g. hyperkalemic ARF, burn injury, muscular dystrophy, spinal cord injury)

Malignant Hyperthermia

- Trismus (masseter muscle spasm) can be a heralding event

Cardiac Arrhythmias

- Bradycardia - parasympathetic and SA node stimulation; especially in children where sympathetic tone is low.
- Cardiac Arrest - successive doses 2-10 minutes apart can cause bradycardia, junctional rhythm, or arrest; always give 2nd dose with 0.4 mg atropine.

Post-operative Myalgias

- Fasciculations have been implicated in causing myalgias.
- Prevented with small defasciculating dose of NDMBs.

Increased ICP, IOP, and intragastric pressure

Non-Depolarizing NMBs

- Mechanism of action by competitive inhibition of ACh at the NMJ.
- Two structural classes:
 1. Benzylisoquinoliniums = “-uriums”
 - Atracurium, Cisatracurium, Mivacurium, Doxacurium, d-Tubocurarine
 - More likely to cause histamine release (d-Tubocurarine >> Atracurium = Mivacurium)
 2. Aminosteroids = “-oniums”
 - Pancuronium, Vecuronium, Rocuronium, Pipecuronium
 - No histamine release
 - May exhibit vagolytic effects (Pancuronium >> Rocuronium >> Vecuronium = Pipecuronium)

Non-Depolarizing NMBs

Short-Acting (onset within 90 sec, offset within 20 minutes)

- Mivacurium = 0.2 mg/kg; metabolized by pseudocholinesterase (but slower than SCh)
- Rapacuronium (off the market due to life-threatening bronchospasm)

Intermediate-Acting (onset within 3 minutes, offset within 30-45 minutes)

- Rocuronium = 0.6 mg/kg (1 mg/kg for RSI with onset similar to SCh); hepatic > renal elimination
- Vecuronium = 0.1 mg/kg; hepatic > renal elimination
- Cisatracurium = 0.2 mg/kg (0.6 mg/kg for RSI); elimination by Hofmann degradation
- Atracurium

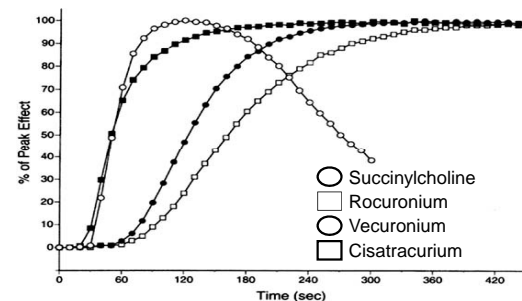
Long-Acting (slow onset, offset ≥ 60 minutes)

- Pancuronium = 0.1 mg/kg; renal > hepatic elimination
- Pipecurium, Doxacurium, d-Tubocurarine

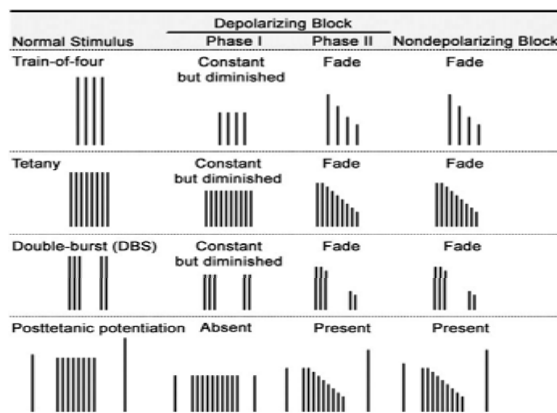
Non-Depolarizing NMBs

- Intubating doses are $2 \times \text{ED}_{95}$
- Precurarization (“defasciculating dose”) with NDMBs reduces the potency and duration of action of SCh.

Time to peak effect for commonly used muscle relaxants



Peripheral Nerve Stimulation



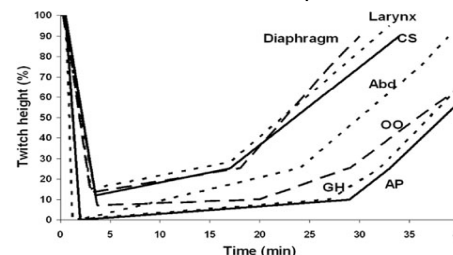
Phase I block is typical for SCh.

Phase II block is typical for NDMBs

SCh can develop a Phase II block at high doses (>6 mg/kg) or with prolonged infusions

Monitoring Neuromuscular Block

- Variability in muscle blockade (most resistant \rightarrow most sensitive): vocal cords > diaphragm > orbicularis oculi (OO) > abdominal muscles > adductor pollicis (AP) > masseter > pharyngeal muscles > extraocular muscles
- Pick one site to monitor (e.g. AP or eyebrow), but know how different muscles respond relative to that site.



Time course after Rocuronium (0.6 mg/kg) at different muscles

- CS = corrugator supercilii (eyebrow)
- Abd = Abdomen
- OO = orbicularis oculi (eyelid)
- GH = geniohyoid (upper airway)
- AP = adductor pollicis (thumb)

Monitoring Neuromuscular Block

Onset of Blockade

- The AP poorly predicts intubating conditions because the diaphragm and laryngeal muscles are MORE resistant to blockade.
- The corrugator supercilii (eyebrow) best predicts laryngeal conditions.

Surgical Relaxation

- The AP is adequate, but is more resistant to recovery than the abdominal muscles.
- Surgeons may complain of “tightness” even though you have no AP twitches.

Recovery from Blockade

- The diaphragm and laryngeal muscles recover first.
- The AP recovers last, so if twitches are present, then the diaphragm can be safely reversed.

Anticholinesterases

- Mechanism of action is by inhibiting acetylcholinesterase thereby increasing the amount of ACh in the NMJ.
- Used as “reversal agents” to counteract NDMBs.
 - Neostigmine, Pyridostigmine, and Edrophonium do not cross the BBB.
 - Physostigmine crosses the BBB (can be used to treat central anticholinergic syndrome/atropine toxicity)
- Anticholinesterases cause vagal side effects (e.g. bradycardia, salivation) by increasing ACh activity at parasympathetic muscarinic receptors; always administer with anticholinergics:
 - We typically use Neostigmine 0.07 mg/kg (~2.5-5 mg) with Glycopyrrolate (0.2 mg per 1 mg Neostigmine)
- Other side effects include nausea and bronchospasm.

Reversal of Neuromuscular Blockade

- NDMB activity is terminated by redistribution away from the NMJ and end-organ metabolism.
- Anticholinesterase “reversal agents” speed up redistribution by increasing ACh levels in the NMJ.
- Assess adequacy for reversal with nerve stimulation:
 - TOF ratio = amplitude of 4th twitch divided by 1st twitch
 - When TOF ratio is 0.7, the single twitch height appears normal, but as many as 70% of receptors are still blocked!
 - Patients can be reversed when ≥ 1 out of 4 twitches is present.
- The gold standard for assessing adequacy of reversal for extubation is 5 seconds of sustained tetany (no fade); other measures include TOF ratio = 0.9 (imperceptible to the eye) or 5 seconds of sustained head lift.

Pearls

- Use Rocuronium for RSI in situations where SCh is contraindicated.
- Consider using Cisatracurium in renal and liver patients (Hofmann degradation).
- Atracurium yields the metabolite “laudanosine”, which can cause CNS stimulation/seizures (but only at high, nonclinical doses)
- Pancuronium is the most renally excreted; causes \uparrow HR, BP, and CO.
- It is important to pair anticholinesterases and anticholinergics based on speeds of onset:
 - Edrophonium (rapid) w/ Atropine
 - Neostigmine (intermediate) w/ Glycopyrrolate
 - Pyridostigmine (slow) w/ Glycopyrrolate

Pearls

- Diseases more RESISTANT to NDMBs:
 - Guillen-Barré (AChR upregulation)
 - Burns (more extrajunctional nAChR)
 - Spinal cord injury
 - CVA
 - Prolonged immobility
 - Multiple sclerosis
- Diseases more SENSITIVE to NDMBs:
 - Myasthenia gravis (fewer AChR)
 - Lambert-Eaton Syndrome (less ACh release)
- Factors ENHANCING block by NDMBs:
 - Volatile anesthetics, aminoglycosides, Mg, IV local anesthetics, CCBs, Lasix, Dantrolene, Lithium, anticonvulsants, Sch, hypokalemia, hypothermia

References

- Donati F and Bevan DR. Neuromuscular blocking agents. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia, 5th ed.* Philadelphia: Lippincott Williams & Wilkins, 2006.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology, 4th ed.* New York: McGraw-Hill Companies, Inc., 2006.
- Schreiber J-U, Lysakowski C, Fuchs-Buder T, et al. 2005. Prevention of succinylcholine-induced fasciculation and myalgia: a meta-analysis of randomized trials. *Anesthesiology*, **103**: 877-84.
- Stoelting RK and Miller RD. *Basics of Anesthesia, 4th ed.* Philadelphia: Churchill Livingstone, 2000.

For a while, one of the surgery residents referred to me as Superman. Not because of anything good, but because I woke his patient up and he emerged a little goofy. He insisted on keeping his arms stretched perfectly straight out in front him, and despite many attempts to get him to relax, he wouldn't put them down. We sat the head of the bed up, thinking that might help, but it just made it more obvious to everyone we drove past on the way to the PACU, with this old guy holding his Superman pose.

I was giving report in the PACU and mistakenly reported that the patient was an otherwise healthy 64 year-old woman. She was awake, and corrected me, noting that she was in fact 44. She was indeed healthy, though.

Difficult Airway Algorithm

STEP 1

Assess the likelihood and clinical impact of basic management problems:

A. Difficult Ventilation

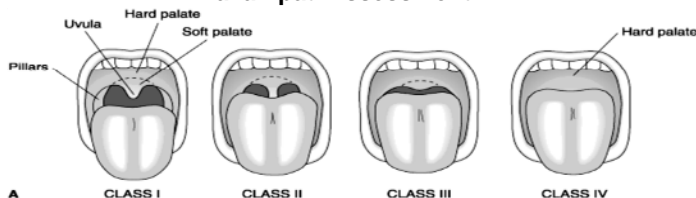
- History of prior difficulty
- Facial hair
- Obesity (BMI > 26 kg/m²)
- History of snoring
- OSA
- No teeth
- Age > 55 years

B. Difficult Intubation

- History of prior difficulty
- Underlying pathology (e.g. laryngeal/tracheal stenosis, epiglottitis, tumors)
- Neck range of motion
- TMJ range of motion
- Thyromental distance
- Mallampati score (see next slide)

STEP 1

Mallampati Assessment



C. Difficulty with patient cooperation

- Age
- Mental capacity
- Level of consciousness

D. Difficulty with tracheostomy

- Obesity
- Facial hair
- Prior ENT surgery
- Prior radiation to neck

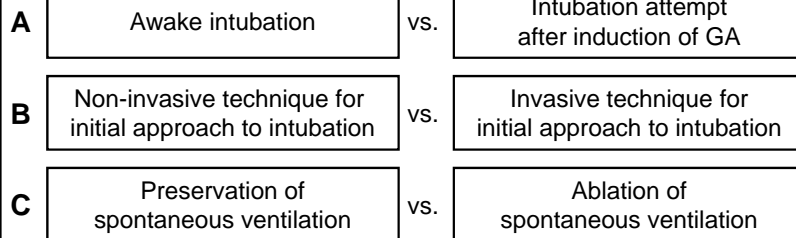
STEP 2

Actively pursue opportunities to deliver supplemental O₂ throughout the process of difficult airway management

- Face mask
- LMA
- FOB swivel adaptor ETT connector
- Patil-Syracuse mask (mask with fiberoptic port)
- FOB side port
- Rigid bronchoscope side port

STEP 3

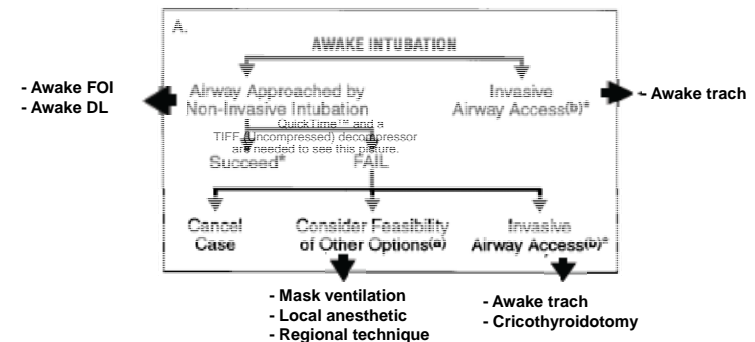
Consider the relative merits and feasibility of basic management choices



STEP 4

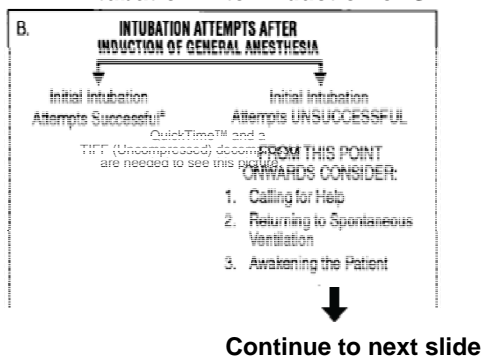
Develop primary and alternate strategies:

Algorithm A: Awake Techniques



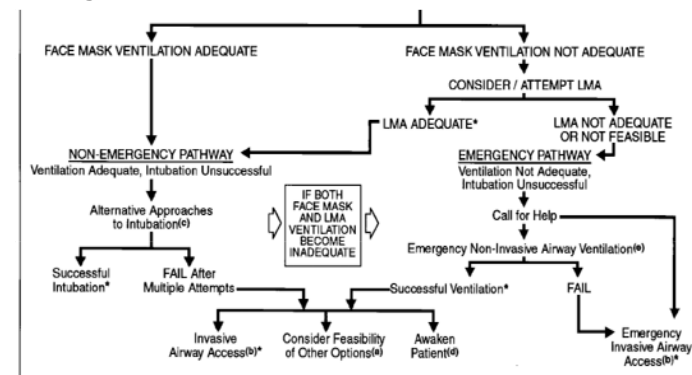
STEP 4

Algorithm B: Intubation After Induction of GA



STEP 4

Algorithm B: Intubation After Induction of GA



Algorithm B

Non-Emergent Pathway

- CALL FOR HELP
- Mask ventilate with cricoid pressure
- Ensure optimal positioning
- Re-attempt DL with different blade
- Consider alternative techniques to secure airway
 - Gum elastic Bougie
 - LMA or intubating LMA
 - Light wand
 - Fiberoptic intubation
 - Retrograde intubation

Algorithm B

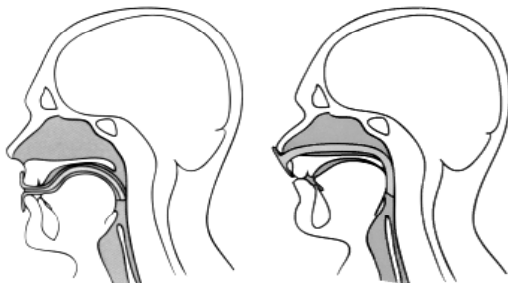
Emergent Pathway

- “Can’t intubate, can’t ventilate”
- CALL FOR HELP
- Emergency Non-Invasive Airway Ventilation
 - Rigid bronch
 - Combitube
 - Transtracheal Jet Ventilation
- Emergency Invasive Airway Ventilation
 - Cricothyroidotomy
 - Surgical trach

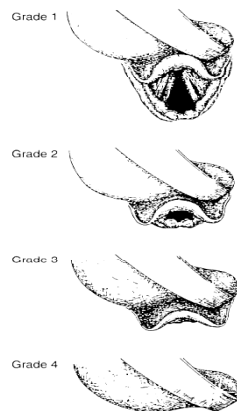
Basics of Airway Management

Oral Airway

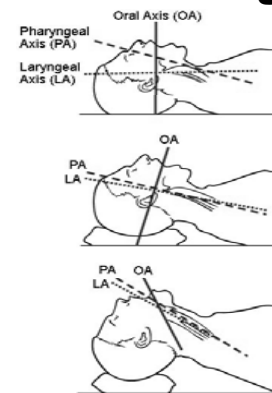
Nasal Airway



Direct Laryngoscopy Views



Positioning and Airway Axis



Head elevation helps to align PA & LA before DL



Ramp up obese patients until tragus is aligned with sternum

Pearls

- CALL FOR HELP
- Always pre-oxygenate (de-nitrogenate)
 - A pre-oxygenated patient can be apneic for 8-10 minutes until desaturation occurs
- The first attempt at DL is the best attempt
- Consider other airway options after 3 attempts at DL
 - Further attempts can cause airway edema and trauma
- Know airway anatomy
- Know pharmacology of anesthetic agents

References

- ASA Task Force on Management of the Difficult Airway. 2003. Practice guidelines for management of the difficult airway. *Anesthesiology*, **98**: 1269-77.
- Benumof JL. 1996. Laryngeal mask airway and the ASA difficult airway algorithm. *Anesthesiology*, **84**: 686-99.
- Collins JS, Lemmens HJM, Brodsky JB, et al. 2004. Laryngoscopy and morbid obesity: a comparison of the "sniff" and "ramped" positions. *Obesity Surgery*, **14**: 1171-5.
- Langeron O, Masso E, Huraux C, et al. 2000. Prediction of difficult mask ventilation. *Anesthesiology*, **92**: 1229-36.
- Gal TJ. Airway Management. In Miller RD (ed), *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- Rosenblatt WH. Airway management. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.
- Rosenblatt WH, Wagner PJ, Ovassapian A, et al. 1998. Practice patterns in managing the difficult airway by anesthesiologists in the United States. *Anesth Analg*, **87**: 153-7.

The first time I had a patient with HIV, I was really nervous about putting in the IV. When I met him in preop, I was relieved that he had really great veins, and I knew he would be really easy. However, I kept missing IV after IV. After the third failed attempt, I finally paged my attending to come over. When he put on the tourniquet, I suddenly realized that that's what I had neglected to do in my previous attempts!

5 minutes after manipulating an NGT that the surgeon insisted wasn't in the stomach (they always say this) when I knew it was because I was getting gastric contents (you always say this), the surgeon complains about a periodic whiff of a foul odor. We all started to notice it. I explained it was probably the gastric contents that leaked out when I was fiddling with the NGT. By the end of the 10 hour case, we pretty much all had some kind of pediatric face mask scent on our masks and everyone that came into our room complained of the smell out in the hall. Then off the came drapes and the horrible truth stared us in the face: The lower body bair hugger was making jerky out of a code brown so massive that it completely filled the void between the patient's legs.

Fluid Management

Eval of Intravascular Volume

- HPI
 - Hypovolemia: vomiting, diarrhea, fever, sepsis, trauma
 - Hypervolemia: weight gain, edema, acute renal failure, liver disease (ascites)
- PE
 - Hypovol: skin turgor, mucous membranes, tachycardia, orthostasis
 - Hypervol: pitting edema, rales, wheezing
- Labs
 - Hypovol: rising Hct, metabolic acidosis, Ur specific gravity > 1.010, Na(Ur) < 10, Osm (Ur) > 450, hypernatremia, BUN:Cr > 10:1

Intraoperative Intravascular Assessment

- **CLINICAL EVALUATION is key!!!**
- Vitals
 - HR, BP, and their changes with positive pressure ventilation
 - Pulse Oximetry: waveform wander from baseline
- Foley Catheter
 - UOP
- Arterial Line
 - Serial ABGs, Hct, electrolytes
 - Commonly used for anticipated blood loss, fluid shifts, prolonged OR time
- Central Venous Pressure
 - Trends often more informative than absolute value
 - Catheter serves as additional central IV access for medications (vasopressors, inotropes) and fluids
 - Consider benefits and risks of placing central line
- Pulmonary Artery pressure
 - Most commonly used in RV dysfunction, PHTN, valvular pathology (AS, MR), LV dysfunction
- Transesophageal Echocardiogram
 - Most commonly used in major heart surgeries and liver transplant
 - Valuable in acute, persistent hemodynamic instability

Fluid Compartments

Males = 60% H₂O by weight
Females = 50% H₂O by weight

	Fluid as % of TBW (%)	Fluid as % of body weight (%)	Volume, in 70 kg male (L)
Intracellular	67	40	28
Extracellular			
- Interstitial	25	13	9
- Intravascular	8	7	5
TOTAL	100%	60%	42 L

TBW = Total Body Water

Q: What is the intravascular volume of a 90 kg male?

A: 90 kg x 7% = 6.3 L

Crystalloids

	Osm (mOsm/L)	Na ⁺ (mEq/L)	Cl ⁻ (mEq/L)	K ⁺ (mEq/L)	Ca ²⁺ (mEq/L)	Lactate (mEq/L)	pH
NS	308	154	154				5.0
LR	273	130	109	4	3	28	6.6

	Advantages	Disadvantages
NS	<ul style="list-style-type: none"> Preferred for diluting pRBCs Preferred in brain injury 	<ul style="list-style-type: none"> May cause hyperchloremic metabolic acidosis Hyperchloremia → low GFR
LR	<ul style="list-style-type: none"> More physiologic Lactate is converted to HCO₃⁻ by liver 	<ul style="list-style-type: none"> Watch K⁺ in renal patients Ca²⁺ may cause clotting with pRBCs

Colloids

Mechanism

- Intravascular volume expansion from increased oncotic pressure

Hetastarch (6% hydroxyethyl starch, HES)

- Hespan (in NS) and Hextend (in LR) solutions
- Solution of highly branched glucose chains (average MW 450 kD)
- Degraded by amylase, eliminated by kidney
- Intravascular t_{1/2} = 25.5 hrs; tissue t_{1/2} = 10-15 days
- Dose: ≤ 20 ml/kg/day (max is roughly 1 L/day)
- Side effects:
 - Can increase PTT (via factor VIII/vWF inhibition), and clotting times
 - Anaphylactoid reactions
 - Can decrease platelet function
- Contraindications: coagulopathy, heart failure, renal failure

Albumin (5% and 25%)

- Derived from donated blood; heat-treated (60 degree C x 10 hrs)
- Use 5% for hypovolemia; 25% for hypovol in Pts with restricted fluid and Na intake
- Min. risk for viral infection (hepatitis or HIV); ? risk of prion transmission
- Expensive; shortages

Colloids

Dextran 40, 70

- Not used at Stanford
- Side effects: anaphylactic reactions (1:3300), antiplatelet activity, renal dysfunction (obstructive), may prolong bleeding time
- Doses > 20 ml/kg/day can interfere with blood typing.

General Indications for Colloids

- Inadequate intravascular volume resuscitation with aggressive crystalloid administration
- Concern for fluid overload with excessive crystalloid (ie. CHF, pulmonary edema, bowel edema)
- Pts with large protein losses (burns)

Crystalloid or Colloid?

	Advantages	Disadvantages
Crystalloid	<ul style="list-style-type: none"> Lower cost Higher UOP 	<ul style="list-style-type: none"> Requires 3-4x more volume for the same hemodynamic effect (due to redistribution) Short IV t_{1/2} (20-30 min) Dilutes plasma proteins → peripheral/pulmonary edema
Colloid	<ul style="list-style-type: none"> Restores IV volume and HD with less volume, less time Longer IV t_{1/2} Maintains plasma oncotic pressure Less cerebral edema Less intestinal edema 	<ul style="list-style-type: none"> Expensive Coagulopathy (dextran > HES) Avoid in hepatic failure Limited by max dose

“Classical” Fluid Management

Maintenance

- “4-2-1 Rule” = 4 ml/kg/hr for the 1st 10 kg, 2 ml/kg/hr for the next 10-20 kg, and 1 ml/kg/hr for each additional kg above 20 kg.

Preexisting Fluid Deficits

- Multiply maintenance requirement by # of hours NPO.
- Give 1/2 over 1st hour, 1/4 over 2nd hour, and 1/4 over 3rd hour

Ongoing Losses

Evaporative and Redistributive (“3rd space”) Losses

- Minimal tissue trauma (e.g. hernia repair) = 0-2 ml/kg/hr
- Moderate tissue trauma (e.g. cholecystectomy) = 2-4 ml/kg/hr
- Severe tissue trauma (e.g. bowel resection) = 4-8 ml/kg/hr

Blood Loss

- EBL = (suction canister - irrigation) + “laps” (100-150 ml each) + 4x4 sponges (10 ml each) + field estimate.
- Replace 1:2 with undiluted pRBCs, 1:1 with colloid, or 3:1 with crystalloid

Urine Output

- Replace 1:1 with crystalloid.

Example

85 kg male s/f colon resection; NPO x 8 hours.

- Maintenance = 125 ml/hr
- Deficit = 125 ml/hr x 8 hrs = 1000 ml
- 3rd Space Losses = 6 ml/kg/hr = 750 ml/hr

	Hour 1	Hour 2	Hour 3	Hour 4	Hour 5
Deficit	500	250	250	--	--
Main	125	125	125	125	125
3 rd Space	--	750	750	750	--
EBL	--	200 (x3)	200 (x3)	200 (x3)	--
UOP	300	100	100	100	100
ml/hr	925	1825	1825	1575	225
TOTAL	925	2750	4575	6150	6375

These calculations assume all EBL replaced 3:1 with crystalloid.

Suggestions for Fluid Management

- Tailor management to patient, surgery, and clinical evaluation
- Consider the calculated “classical” fluid management
- Maintain stable VS, UOP > 0.5 ml/kg/hr, adequate CVP
- Use a balanced approach
 - Typically start with NS or LR
 - If Pt requires >2-3L fluids, consider alternating NS and LR
 - Consider colloid for persistent hypotension despite adequate crystalloid administration.
- Type and Cross for pBRC and other blood products prior to surgery if anticipating significant blood loss (ie. trauma, coagulopathy)

Liberal vs. Restrictive Management

Consequences of Excessive Peri-op Fluid Administration

- Increased mortality and length of ICU/hospital stay
- Increased myocardial morbidity
- Increased pulmonary, periorbital, and gut edema
- Increased PONV and delayed return of GI function
- Decreased hematocrit and albumin
- Decreased wound/anastomosis healing (edema)

Suggestions for Rational Fluid Management

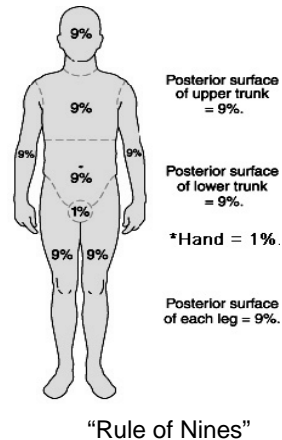
- Consider the calculated “classical” (i.e. liberal) fluid management, but use good clinical judgment.
- Tailor management to patient, surgery, and clinical picture.
- Maintain UOP > 0.5 ml/kg/hr, adequate CVP, and stable VS.
- Use balanced fluid therapy: use crystalloid for maintenance, replace EBL 1:1 with colloid.
- Consider conservative replacement of 3rd space losses or UOP unless VS unstable.

Burns

- Increased evaporative losses.
- H₂O, electrolytes, and protein shift from normal to burned tissue, causing intravascular hypovolemia.
- Volume to infuse is calculated by the Parkland Formula

Parkland Formula

- Volume = %BSA x 4 ml/kg x kg
- Give 1/2 over the 1st 8 hours.
- Give 1/2 over the next 16 hours.
- Replace with LR.
- %BSA is determined by the "Rule of Nines"



Intraoperative Oliguria

1. Pre-renal (decreased renal perfusion)

- Hypovolemia
- Decreased CO (LV dysfunction, valvular disease)
- Decreased MAP

2. Post-renal (post-renal obstruction)

- Foley kinked, clogged, displaced, or disconnected
- Surgical manipulation of kidneys, ureters, bladder, or urethra

3. Renal

- Neuroendocrine response to surgery (i.e. activation of renin-angiotensin-aldosterone system)

Treatments

- Increase renal perfusion: fluids (bolus vs increased maintenance rate), vasopressors/inotropes, or Lasix
- Relieve obstruction: check Foley; consider IV dyes (e.g. indigo carmine, methylene blue) for patency of ureters (usually in Urology cases)

References

- Holte K, Sharrock NE, and Kehlet H. 2002. Pathophysiology and clinical implications of perioperative fluid excess. *Br J Anaesth*, **89**: 622-32.
- Joshi GP. 2005. Intraoperative fluid restriction improves outcome after major elective gastrointestinal surgery. *Anesth Analg*, **101**: 601-5.
- Kaye AD and Kucera IJ. Intravascular fluid and electrolyte physiology. In Miller RD (ed), *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- McKinlay MB and Gan TJ. Intraoperative fluid management and choice of fluids. In Schwartz AJ, Matjasko MJ, and Otto CW (eds), *ASA Refresher Courses in Anesthesiology*, **31**: 127-37. Philadelphia: Lippincott Williams & Wilkins, 2003.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill Companies, Inc., 2006.

The first time I emptied urine, it sprayed all over my scrubs. Apparently it's better to aim the spout downwards into the empty bottle before you release the clamp, not up at yourself.

Transfusion Therapy

Type and Screen/Crossmatch

Type and Screen (takes 30-120 min, lasts 72 hr)

- ABO-Rh typing and antibody screen
 - Recipient serum + type O RBCs for presence of A or B antibodies - no agglutination = negative screen
 - If antibody screen is positive the serum is tested further
 - Recipient RBCs for presence of A or B antigens

Type and Crossmatch (if T&S negative takes 30-60 min)

- Immediate phase: recipient serum + donor cells test for recipient Ab to donor (5 minutes)
- Incubation phase: incubate products from first test to look for incomplete recipient Ab to donor ie. Rh system
- Indirect Antiglobulin test: antiglobulin serum to products of first two tests to look for incomplete recipient Ab to Rh, Kell, Duffy, and Kidd

Packed Red Blood Cells

Definition, Use, & Storage

- Single donor; volume 250-300 ml with Hct ~70%.
- 1 unit pRBCs \uparrow adult Hgb ~1 g/dl or Hct ~3%.
- 10 ml/kg PRBC \uparrow Hct 10%
- Stored at 4°C in CPD (21 days), CPDA (35 days), or Adsol (42 days).
- CPDA:
 - Citrate (anticoagulant) - also binds iCa
 - Phosphate (buffer)
 - Dextrose (energy source)
 - Adenosine (precursor to ATP synthesis)

Packed Red Blood Cells

Indications (ASA Guidelines)

1. H/H < 6/24 in young, healthy patients
2. Usually unnecessary when H/H > 10/30 g/dl
3. At Hgb 6-10 g/dl, the decision to transfuse is based on:
 1. ongoing indications of organ ischemia
 2. potential or ongoing blood loss
 3. volume status
 4. risk factors for complications of inadequate O₂.

Note:

1. Solutions incompatible with pRBC:
LR (theoretical clot formation due to calcium
D5W, plasmanate, 0.2% saline (hemolysis)

Platelets

Definition, Use, & Storage

- Platelet Concentrate (PC)
 - Platelets from one donated unit, vol = 50-70 ml; ↑ plt ~5000-10,000.
 - “6-pack” = 6 pooled PCs; rarely used anymore
- Apheresis Unit
 - Platelets from a single donor; vol = 200-400 ml; ↑ plt ~50,000.
- Can give ABO-incompatible platelets, Rh tested only
- Stored at room temperature for ≤5 days.

Indications (ASA Guidelines)

1. Rarely when plt > 100,000
2. Usually when plt < 50,000 (spontaneous bleed at < 20K)
3. When plt 50-100,000, based on risk of bleeding
4. With platelet dysfunction (e.g. CPB, plt inhibitors)

Fresh Frozen Plasma

Definition, Use, & Storage

- Fluid portion from whole blood
- Contains all coagulation factors (except platelets)
- 1 unit increases clotting factors 2-3%
- Use ABO-compatible; Rh-incompatible is OK
- Stored frozen; takes 30 min to thaw; use within 24 hrs of thawing

Indications (ASA Guidelines)

1. Urgent reversal of Coumadin
2. Correction of known factor deficiency
3. Correction of 1) microvascular bleeding with INR > 1.5, 2) INR > 2, or 3) PTT > 2x normal
4. During massive transfusion (before lab results available)
5. Heparin resistance (i.e. antithrombin III deficiency) in patients requiring heparinization.

Cryoprecipitate

Definition

- Fraction of plasma that precipitates when FFP is thawed.
- Contains Factors VIII, XIII, I (fibrinogen), and fibronectin
- 1 unit contains ~5X more fibrinogen than 1 unit FFP.
- Use within 4-6 hours after thawed if want to replace Factor VIII

Indications (ASA Guidelines)

1. Rarely when fibrinogen >150 mg/dl
2. When fibrinogen <100 mg/dl with microvascular bleeding
3. During massive transfusion when fibrinogen level not available
4. Bleeding patients with vWF disease
5. Congenital fibrinogen deficiency

Equations

Arterial O₂ Content

$$\begin{aligned}
 C_aO_2 &= O_2\text{-Hb} + \text{Dissolved } O_2 \\
 &= (Hb \times 1.36 \times S_aO_2/100) + (P_aO_2 \times 0.003) \\
 &= (15 \times 1.36 \times 100\%) + (100 \times 0.003) \\
 &\approx \underline{20 \text{ cc } O_2/\text{dl}}
 \end{aligned}$$

Allowable Blood Loss

$$\text{ABL} = \frac{[\text{Hct (start)} - \text{Hct (allowed)}] \times \text{EBV}}{\text{Hct (start)}}$$

Volume to Transfuse

$$\text{Volume} = \frac{[\text{Hct (desired)} - \text{Hct (current)}] \times \text{EBV}}{\text{Hct (transfused blood)}}$$

Estimated Blood Volume (ml/kg)

Preemie	100
Term	90
< 1 year	80
1-6 years	75
Male	70
Female	65
Obese	≤60

Transfusion-Related Infections

Viral

- CMV >1:100
 - Hepatitis B 1 in 220,000
 - Hepatitis C 1 in 1,600,000
 - HIV 1 in 2,000,000
- (Figures based on 2000-2001 estimated risk)

Bacterial

- Most common with platelets (1:2000) due to their storage in dextrose at room temperature.
- pRBCs not a major source (1:500,000) due to their storage at 4°C, but Yersinia is most likely organism.

Blood is screened for HCV, HBV core Ab, HIV-1, HIV-2, HTLV, syphilis

Transfusion Reactions

Febrile Non-Hemolytic Reaction

- Benign; occurs with 0.5-1% of transfusions
- R/O acute hemolytic reaction
- Treatment: Tylenol, Benadryl, supportive care

Allergic/Anaphylactic Reaction

- Occurs within minutes; life-threatening
- Signs/Symptoms: shock, angioedema, ARDS
- Treatment: D/C blood, fluids, Epi, antihistamines, ACLS

Acute Hemolytic Reaction

- Due to ABO incompatibility
- Symptoms (fever, chills, flank pain) masked by GA; watch for hypotension & brown urine; monitor for ARF and DIC.
- Treatment: D/C blood, maintain alkaline UOP (NaHCO_3^- , mannitol, Lasix), supportive care.

Transfusion-Related Acute Lung Injury (TRALI)

TRALI

- An acute RDS that occurs ~4 hours after transfusion.
- Incidence: 1 in 1120 (but underreported)
- Mortality 5-10%
- Due to plasma-containing products (platelets and FFP > pRBCs)
 - usually donor origin antibodies to leukocytes
- Signs & symptoms: Dyspnea, hypoxemia, hypotension, fever, pulmonary edema.
- Diagnosis of exclusion: first R/O sepsis, volume overload, and cardiogenic pulmonary edema
- Treatment: supportive care, similar to ARDS (O_2 , mechanical ventilation, volume)
- TRALI is usually self-limited and resolves within 48 hours.

Massive Transfusion

Definition

- Acute administration of greater than 1 blood volume (~10 units) in 24 hours.
- At Stanford, calling the blood bank for the Massive Transfusion Guideline (MTG) will get you 6 pRBCs, 4 FFP, and 1 unit of platelets.

Consequences

1. Hypothermia
 - Blood products are stored cold - use a fluid warmer!
2. Coagulopathy
 - a. Dilutional thrombocytopenia
 - Platelet count likely <100,000 after ~10 units pRBCs
 - b. Dilutional coagulopathies
 - ↓ Factors V & VIII ("labile factors") in stored blood
 - Hypofibrinogenemia

Massive Transfusion

Consequences

3. Citrate Toxicity

- Citrate is in CPDA storage solution as a Ca^{2+} chelator.
- Massive transfusion can cause an acute hypocalcemia.
- Binds magnesium as well causing hypomagnesemia

4. Acid-Base Abnormalities

- At 21 days, stored blood has pH <7.0, due mostly to CO_2 production, which is rapidly blown off after transfusion.

5. Hyperkalemia

- K^+ moves out of pRBCs during storage.
- If EKG changes occur, stop transfusion and treat hyperkalemia.

6. Impaired O_2 -Carrying Capacity (!?!)

- 2,3-DPG decreases in stored blood, causing a left-shifted O_2 -Hb dissociation curve.

References

- ASA Task Force on Perioperative Blood Transfusion and Adjuvant Therapies. 2006. Practice guidelines for perioperative blood transfusion and adjuvant therapies. *Anesthesiology*, **105**: 198-208.
- Goodnough LT. 2003. Risks of blood transfusion. *Crit Care Med*, **31**: S678-86.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill Companies, Inc., 2006.

Actual conversation in a case:

Nameless neurosurgeon (NN) "What's the MAP"

Anesthesia Attending (AA) "65"

NN "Too high. Make it 55"

45 seconds later

AA "The MAP is now 55"

NN "That's way too low. Make it 65 again"

Moral = sometimes you can just never win.

I was about to infiltrate a pt's arm with lidocaine for an IV, when both the patient and I both realized that he had an anaphylactic allergy to lidocaine! He recoiled in fear. I then proceeded to blow his IV without lidocaine.

Hypoxemia

Causes of Hypoxemia

	$P_a\text{CO}_2$	A-a Gradient	DLCO	Corrects w/ 100% $F_i\text{O}_2$?
Low $F_i\text{O}_2$	Normal	Normal	Normal	Yes
Hypoventilation	↑	Normal	Normal	Yes
Diffusion Impairment	Normal	↑	↓	Yes
Shunt	Normal	↑	Normal	No
V/Q Mismatch	Normal / ↑	↑	Normal	Yes

Shunt: perfusion without ventilation ($V/Q=0$); see ↓ $p\text{O}_2$

Dead Space: ventilation without perfusion ($V/Q=\infty$); see ↑ $p\text{CO}_2$

Equations

Alveolar-arterial (A-a) Gradient

$$P_{(A-a)}\text{O}_2 = P_A\text{O}_2 - P_a\text{O}_2$$

Alveolar Gas Equation

$$\begin{aligned}
 P_A\text{O}_2 &= F_i\text{O}_2 (P_{\text{atm}} - P_{\text{H}_2\text{O}}) - (P_a\text{CO}_2 / 0.8) \\
 &= 0.21 (760 - 47) - (40 / 0.8) \\
 &\approx \underline{100 \text{ mm Hg}}
 \end{aligned}$$

Normal A-a Gradient:

- < 10 mm Hg ($F_i\text{O}_2 = 0.21$)
- < 60 mm Hg ($F_i\text{O}_2 = 1.00$)
- < (age / 4) + 4
- a/A ratio > 0.75

Normal $P_a\text{O}_2$:

- 103 - age/3

Causes of Hypoxemia

1. Low $F_i\text{O}_2$

- Altitude
- Hypoxic $F_i\text{O}_2$ gas mixture

2. Hypoventilation

- Drugs (opioids, BZDs, barbiturates)
- Chest wall damage
- Neuromuscular diseases
- Obstruction (e.g. OSA, upper airway compression)

3. Diffusion Impairment

- Increased diffusion pathway (e.g. pulmonary edema, fibrosis)
- Decreased surface area (e.g. emphysema, pneumonectomy)
- Decreased rate of O_2 -Hb association (e.g. high CO, anemia, PE)

Causes of Hypoxemia

4. Shunt (i.e. perfusion w/o ventilation; $V/Q = 0$)

- Congenital (e.g. ASD, VSD, PDA), or AVM
- ARDS, pneumonia, atelectasis

5. V/Q Mismatch

- Often multifactorial
- COPD, ILD, PE
- Decreased CO (e.g. MI, CHF)

6. Mixed Process

- Hypoxemia is often due to multiple causes.
- Example: A tourist with COPD is visiting Denver, overdoses on heroin, now s/p MVA with chest wall trauma, pulmonary hemorrhage, Hct = 15%, and LV contusion. What is the cause of hypoxemia?

Hypoxemia in the OR

Take a systematic approach to the diagnosis and treatment of hypoxemia in the OR!

Suggestion: *Alveoli → Machine*

1. Listen to the lungs

- Atelectasis
- Pulmonary edema
- Bronchoconstriction
- Mucus plug or secretions
- Right mainstem ETT
- Pneumothorax
- Esophageal intubation

2. Check ETT

- Cuff deflation
- Kinked/bitten ETT
- Extubation

Hypoxemia in the OR

3. Check circuit

- ETT disconnect
- Circuit disconnect

4. Check machine

- Inspiratory & expiratory valves
- Bellows
- Minute ventilation
- F_iO_2
- Pipeline & cylinder pressures

5. Check monitors to confirm (you will probably do this 1st!)

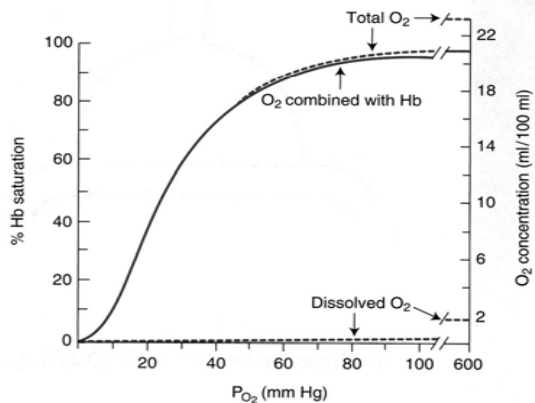
- Pulse oximeter waveform
- Gas analyzer

Management of Hypoxemia

Assuming proper oximeter function, placement, and waveform:

- Place patient on 100% O_2 .
- Perform recruitment maneuver and/or add PEEP.
- Confirm ETT placement by auscultation, bilateral chest rise, and FOB if necessary.
- Suction airway.
- Consider cardiovascular causes and restore volume, RBCs and/or cardiac output.

O₂-Hb Dissociation Curve



Useful “anchor” points:

S _{O2}	P _{O2} (mm Hg)
50%	27
75%	40
97%	100

Note:
P₅₀ ≈ 27 mm Hg

O₂-Hb Curve Shifts

Right Shift

(lower affinity for O₂ = increased unloading at tissues)

- Acidosis
- Hyperthermia
- Hypercarbia
- Increased 2,3-DPG
- Sick Cell Hb
- Pregnancy
- Volatile anesthetics
- Chronic anemia

Left Shift

(higher affinity for O₂ = decreased unloading at tissues)

- Alkalosis
- Hypothermia
- Hypocarbia
- Decreased 2,3-DPG
- CO-Hb
- Met-Hb
- Sulf-Hb
- Fetal Hb
- Myoglobin

Factors Affecting Tissue Oxygenation

- Hb concentration
- O₂ Saturation
- Cardiac Output
- O₂ Consumption
- O₂-Hb Affinity (P₅₀)
- Dissolved O₂ in plasma (little effect)

See “Equations” for a mathematical explanation of these factors.

Equations

Arterial O₂ Content

$$\begin{aligned}
 C_aO_2 &= O_2\text{-Hb} + \text{Dissolved } O_2 \\
 &= (Hb \times 1.36 \times S_aO_2/100) + (P_aO_2 \times 0.003) \\
 &= (15 \times 1.36 \times 100\%) + (100 \times 0.003) \\
 &\approx \underline{20 \text{ cc } O_2/\text{dl}}
 \end{aligned}$$

Mixed Venous O₂ Content

$$\begin{aligned}
 C_vO_2 &= O_2\text{-Hb} + \text{Dissolved } O_2 \\
 &= (Hb \times 1.36 \times S_vO_2/100) + (P_vO_2 \times 0.003) \\
 &= (15 \times 1.36 \times 75\%) + (40 \times 0.003) \\
 &\approx \underline{15 \text{ cc } O_2/\text{dl}}
 \end{aligned}$$

Equations

O₂ Delivery

$$\begin{aligned} \text{DO}_2 &= \text{CO} \times \text{C}_a\text{O}_2 \\ &= 5 \text{ L/min} \times 20 \text{ cc O}_2/\text{dl} \\ &\approx 1 \text{ L O}_2/\text{min} \end{aligned}$$

O₂ Consumption (Fick Equation)

$$\begin{aligned} \text{VO}_2 &= \text{CO} \times (\text{C}_a\text{O}_2 - \text{C}_v\text{O}_2) \\ &= 5 \text{ L/min} \times 5 \text{ cc O}_2/\text{dl} \\ &\approx 250 \text{ cc O}_2/\text{min} \end{aligned}$$

O₂ Extraction Ratio

$$\begin{aligned} \text{ER}_{\text{O}_2} &= (\text{VO}_2 / \text{DO}_2) \times 100 \\ &= 250 / 1000 \\ &\approx 25\% \text{ (normal 22-30\%)} \end{aligned}$$

Other Concepts

Diffusion Hypoxia = low P_AO₂ as a result of breathing air, in combination with the washout of N₂O into the alveoli, upon termination of an anesthetic.

Absorption Atelectasis = the tendency for airways to collapse if proximally obstructed; poorly soluble N₂ normally stents alveoli open, but patients on 100% O₂ have greater tendency toward atelectasis.

Bohr Effect = a property of Hb in which increasing CO₂, temperature, and acidosis promote decreased O₂-Hb affinity (i.e. right-shift of O₂-Hb curve).

Haldane Effect = a property of Hb in which O₂ promotes dissociation of CO₂ from Hb to the plasma (e.g. as when venous blood enters the lungs).

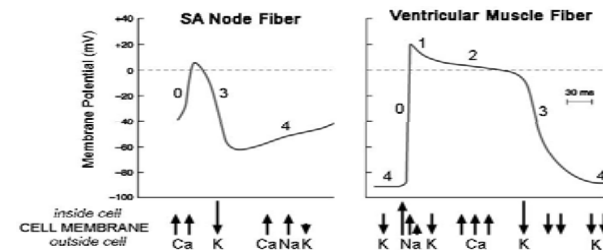
References

- Gaba DM, Fish KJ, and Howard SK. *Crisis Management in Anesthesiology*. Philadelphia: Churchill Livingstone, Inc., 1994.
- West JB. *Respiratory Physiology: The Essentials*, 7th ed. Philadelphia: Lippincott Williams & Wilkins, 2005.
- West JB. *Pulmonary Pathophysiology: The Essentials*, 6th ed. Philadelphia: Lippincott Williams & Wilkins, 2003.

In one of my first days of residency (I was at the Valley, where there are 5 or 6 different kinds of anesthesia machines), it took me about 10 minutes in the morning to find the power button for the ventilator. I felt pretty dumb. The problem ended up being that I had a towel draped over the tray and it was obscuring the otherwise direct view of the right button. But it's a humbling reminder that our job is a mix of complex physiology / pharmacology / etc. and very practical, mundane details. You can master all the ventilator physiology you want, but it won't do you much good if you can't turn the ventilator on.

Electrolyte Abnormalities

Cardiac Action Potentials



Phase	Phase Name	SA Node Fiber	Ventricular Muscle Fiber
0	Rapid Upstroke	Slow inward I_{Ca}	Fast inward I_{Na}
1	Early Rapid Repolarization	—	Inactivation of I_{Na} Start outward I_K
2	Plateau	—	Slow inward I_{Ca} = Outward I_K
3	Final Rapid Repolarization	Outward I_K	Inward I_{Ca} < Outward I_K
4	Diastolic Depolarization/ Resting Potential	Slow inward I_{Ca} Slow inward I_{Na} Outward I_K (minimal)	Outward I_K

Hyperkalemia

Definition

- Mild $K^+ = 5.5-6.5$ mEq/L
- Moderate $K^+ = 6.5-8$ mEq/L
- Severe $K^+ > 8$ mEq/L

Contributing Factors

Preoperative

- Renal disease
- Drugs (ACEI, NSAIDs, spironolactone, Digoxin, β -blockers)

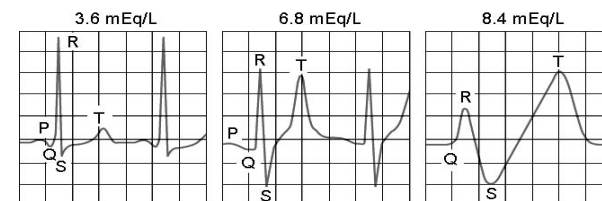
Intraoperative

- Succinylcholine: acute increase of 0.5-1 mEq/L
- Acidosis
- Transfusions
- Hemolysis
- Rhabdomyolysis (tourniquet), trauma

Hyperkalemia

Signs & Symptoms

- EKG changes (usually appear at $K^+ > 6.5$ mEq/L)
 1. Peaked T waves
 2. Flattened P waves & prolonged PRI
 3. Widened QRS \rightarrow sinusoidal QRS
 4. ST elevation
 5. VF arrest & asystole
- Weakness



Hyperkalemia

Treatment

- Calcium
 - Ca gluconate (peripheral)
 - Ca chloride (central)
- Bicarbonate (NaHCO_3)
- Insulin (10-15 Units)
- Glucose (25 g)
- Kayexalate (PO/PR)
- Dialysis

Mnemonic: C BIG K

Hyperkalemia

Anesthetic Considerations

- Consider cancelling elective cases if $\text{K}^+ > 5.5$
- Consider alternative to succinylcholine
- EKG monitoring
- Avoid hypoventilation (respiratory acidosis)
- Treat acidosis
- Use NS instead of LR
- Monitor for increased sensitivity to muscle relaxants.

Hypokalemia

Definition

- Mild $\text{K}^+ = 3.1-3.5 \text{ mEq/L}$
- Moderate $\text{K}^+ \leq 3 \text{ mEq/L}$ with PACs
- Severe $\text{K}^+ < 3 \text{ mEq/L}$ with PVCs

Contributing Factors

Preoperative

- GI losses (NGT, N/V, Diarrhea)
- Lasix, RTA
- Magnesium deficiency

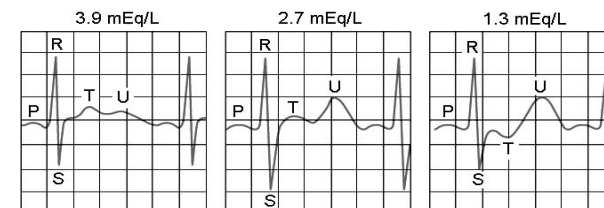
Intraoperative

- Alkalosis (both metabolic and respiratory)
- Insulin therapy
- Hypothermia

Hypokalemia

Signs & Symptoms

- EKG changes
 1. Flattened/inverted T wave
 2. U waves
 3. ST depression
- Arrhythmias
 1. PACs, PVCs
 2. SVTs (esp. A Fib/A flutter)
- Metabolic alkalosis
- Autonomic lability
- Enhanced response to muscle relaxants
- Weakness, ↓DTRs
- Ileus
- Digoxin toxicity



Hypokalemia

Treatment

Chronic hypokalemia = total body K^+ depletion (1 mEq/L decrease = 300-600 mEq total body deficit)

- Peripheral IV - 10 mEq/hr
- Central IV - 10-20 mEq/hr
- Life-threatening - 5-6 mEq bolus

Acute hypokalemia = likely a redistribution phenomenon

- Reverse underlying cause

Hypokalemia

Anesthetic Considerations

- Consider cancelling elective cases if $K^+ < 3-3.5$ mEq/L (based on chronicity of deficit).
- EKG monitoring
- KCl replacement if arrhythmias develop
- Avoid hyperventilation (respiratory alkalosis)
- Consider reducing dose of muscle relaxant 25-50%

Hypercalcemia

Contributing Factors

- Hyperparathyroidism
- Malignancy (especially lung, ENT, GU, GYN, and multiple myeloma)
- Immobilization
- ARF
- Drugs (thiazide diuretics, lithium)

Signs & Symptoms

- EKG changes(short QT)
- Hypertension
- Polyuria

Treatment

- Hydration + Lasix diuresis
- Dialysis

Hypercalcemia

Anesthetic Considerations

- Consider cancelling elective cases
- Avoid acidosis (reduces Ca^{2+} -albumin binding)
- Check serial K^+ and Mg^{2+}

Hypocalcemia

Contributing Factors

Preoperative

- Hypoparathyroidism
- Renal failure (decreased Vitamin D)
- Sepsis
- Magnesium deficiency (decreased end-organ response to PTH)

Intraoperative

- Alkalosis (increased Ca^{2+} -albumin binding)
- Massive pRBC transfusion (due to citrate binding)
- Drugs (heparin, protamine, glucagon)

Signs & Symptoms

- EKG (prolonged QT, bradycardia)
- Hemodynamics (vasodilation, hypotension, decreased myocardial contractility, LV failure)
- Respiratory (laryngospasm, stridor, bronchospasm, respiratory arrest)
- Neuro (cramps, tetany, \uparrow DTRs, perioral numbness, seizures, Chvostek's sign, Trousseau's sign)

Hypocalcemia

Treatment

- Calcium gluconate - 1 g = 4.5 mEq elemental Ca^{2+} (give via peripheral or central IV)
- Calcium chloride - 1 g = 13.6 mEq elemental Ca^{2+} (give via central IV)
- Do NOT give Ca^{2+} and NaHCO_3 together in the same IV - it will precipitate!
- Replace magnesium

Anesthetic Considerations

- EKG monitoring
- Avoid alkalosis
- Monitor paralysis with muscle relaxants
- Monitor iCa with transfusions

Hypermagnesemia

Contributing Factors

- Renal failure
- Hypothyroidism
- Iatrogenic (tocolysis)

Signs & Symptoms

- EKG (widened QRS, prolonged PRI, bradycardia)
- Hemodynamics (vasodilation, hypotension, myocardial depression)
- Neuro (\downarrow DTRs, sedation, weakness, enhanced neuromuscular blockade)

Treatment

- Hydration + Lasix diuresis
- Ca^{2+} administration
- Diuresis

Anesthetic Considerations

- EKG monitoring
- Consider reducing dose of muscle relaxants 25-50%

Hypomagnesemia

Contributing Factors

- GI/Renal losses
- β -agonists (cause intracellular shift)
- Drugs (diuretics, theophylline, aminoglycosides, amphotericin B, CSA)

Signs & Symptoms

- Usually asymptomatic alone, but symptomatic in combination with induced hypokalemia, hypocalcemia, and hypophosphatemia
- EKG (prolonged QT, PACs, PVCs, and A Fib)
- Neuro (neuromuscular excitability, AMS, seizures)

Treatment

- Replace with MgSO_4 to $[\text{Mg}^{2+}] > 2 \text{ mg/dl}$
- Watch for hypotension & arrhythmias with rapid administration!

Anesthetic Considerations

- EKG monitoring
- Check for coexistent electrolyte deficiencies.

References

- Kaye AD and Kucera IJ. Intravascular fluid and electrolyte physiology. In Miller RD (ed), *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill Companies, Inc., 2006.
- Prough DS, Wolf SW, Funston JS, and Svensén CH. Acid-base, fluids, and electrolytes. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.

I was in the middle of a long, stable but tedious endometriosis case in the ASC. I tried to open my next vial of dilaudid and bam! It shattered in my hand and I had 2mg of dilaudid dripping down my fingers. Not wanting to be pegged as a CA-1 with a drug problem, I quietly called the pharmacy to ask them how to document the incident. The discussion took about a minute or so, and when I hung up, I realized the attending surgeon had stopped the case and was staring at me, as was everyone else in the room. He told me he gets "easily distracted" and so he was patiently waiting until I was off the phone!

During the middle of a straightforward case I was drawing up my drugs for the next case. I dropped the propofol vial but after inspection nothing was damaged. I proceeded to inject air into the vial making it easier to draw up. Needless to say it exploded on me.....and the sterile operative field. Bummer.

CSI tip: In July, keep your eyes peeled for distinctive splatter patterns of white stuff on new residents' scrubs, badges, or other paraphernalia. It is a sign that they, too, have been sprayed with either Propofol or Kefzol while trying to draw up a syringe. The needle tip has to stay inside the vial.

CSI tip: don't believe it if another CA1 has a BandAid on their finger or hand and they tell you they cut themselves in the kitchen or have a paper cut. Odds are they stabbed themselves with a needle drawing up drugs in the morning. Hope it was clean!

Hypothermia & Shivering

Definition and Measurement

- Hypothermia is defined as a core body temperature less than 36 degrees C
- Temperature is measured from:
 - Nasopharynx (accurately reflects core temp, but can cause epistaxis)
 - Tympanic Membrane (reflects brain temp, but can cause perforation of ear drum)
 - Esophagus
 - Bladder
 - Rectum (slow response to changes in core temp, contraindicated in neutropenic pt, fistula, etc.)
 - Skin (variable accuracy depending on skin perfusion)
 - Thermistor of Pulmonary Artery Catheter

Thermoregulation

Afferent Thermal Sensing

- Thermal inputs travel along A-delta (cold) and C fibers (warm) via the spinothalamic tract.
- Input comes from the skin, deep abdominal & thoracic tissues, spinal cord, brain, and hypothalamus (roughly 20% each).

Central Control

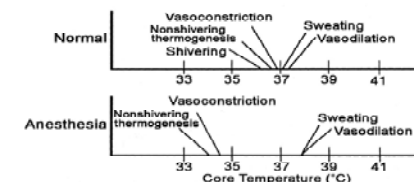
- Thermal inputs are “preprocessed” at numerous levels within the spinal cord and brainstem.
- Modulated by NE, DA, 5-HT, ACh, PGE, and neuropeptides.
- The preoptic-anterior hypothalamus is the central autonomic thermoregulatory center.

Efferent Responses

- Behavioral responses (shelter, clothing, voluntary movement, etc) are most important and are determined by skin temperature.
- Autonomic responses (skin vasomotor activity, nonshivering thermogenesis, shivering, and sweating) are ~80% determined by core temperature.

Interthreshold Range

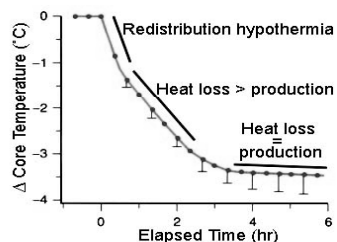
- Interthreshold Range = tight thermoregulatory range between cold-induced and warm-induced responses, usually ~0.2°C.
- General anesthesia inhibits thermoregulation and increases the interthreshold range ~20-fold, to ~4°C.
- Regional anesthesia inhibits thermoregulation to lower half of body, increasing the range ~4-fold, to ~0.8°C.



Development of Hypothermia

Anesthetic-impaired thermoregulation

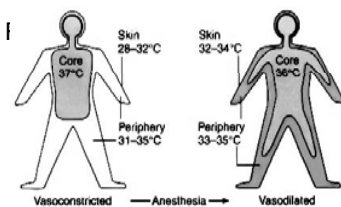
1. Redistribution hypothermia
2. Heat loss > heat production
3. Heat loss = heat production (steady-state heat balance)



Heat transfer to cold OR (in order of importance)

1. Radiation
2. Convection
3. Evaporation
4. Conduction

Redistribution Hypothermia



Benefits of Hypothermia

- Tissue metabolic rate decreases ~8% per 1°C decrease in body temperature.
- CNS protection from ischemic and traumatic injuries.
- Improves neurologic outcomes after cardiac arrest.
- Some protection against malignant hyperthermia.
- Cardiac Protection as decreased metabolic and O₂ requirement.

Consequences of Hypothermia

- Increased myocardial morbidity (3x)
- Impaired coagulation (especially platelets), increased blood loss, & increased transfusion rates
- Increased infection rate (3x)
- Prolonged duration of drug action, delayed emergence
- Left-shifts O₂-Hb curve
- Increased SVR
- Difficulty monitoring patient (e.g. S_pO₂)
- Delays wound healing & jeopardizes grafts
- Altered mental status
- Increased sympathetic activity/stress response
- Increased postoperative shivering
- Prolonged PACU stay

Warming Strategies

Prevention of hypothermia is more effective than treatment!

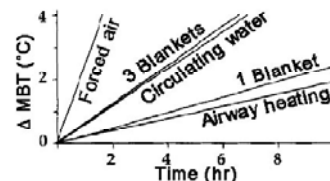
Active Warming

- Forced air (Bair Hugger)
- Circulating warm H₂O pad
- Radiant heat lamps
- IVF warmer
- Airway heating & humidification
- Warm the OR temperature

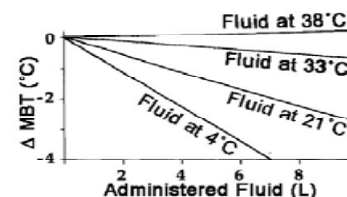
Passive Insulation (not as effective)

- Cotton blankets
- Surgical drapes
- Space blanket (silver plastic)

Effect of Warming Strategies



Effect of IVF Warming



Etiology of Postop Shivering

Intraoperative hypothermia (duh!)... *however...*

- Shivering does NOT always occur in hypothermic patients, and...
- Shivering DOES occur in normothermic patients

Other possible etiologies:

- Recovery from volatile anesthetics
- Pain may facilitate shivering-like tremor
- Fever increases the thermoregulatory set point causing shivering in normothermic patients.

Consequences of Shivering

- Increased O₂ consumption
 - Can be up to a 400-500% increase
- Increased CO₂ production and V_E
- Increased incidental trauma
- Increased intraocular and intracranial pressures
- Uncomfortable and/or painful
- Stresses wound edges
- Disrupts monitoring (e.g. NIBP, EKG, S_pO₂)

Rates of MI do NOT correlate with shivering!

Treatment of Shivering

1. Skin surface warming and passive insulation
2. Pharmacologic:
 - Meperidine 12.5-25 mg IV
 - Muscle relaxants (only in asleep, ventilated patients)

References

- De Witte J, and Sessler DI. 2002. Perioperative shivering: physiology and pharmacology. *Anesthesiology*, **96**: 467-84.
- Sessler DI. Temperature monitoring. In Miller RD (ed), *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- Sessler DI. Mild perioperative hypothermia. *NEJM*, **336**: 1730-7.
- Morgan, GE. *Clinical Anesthesiology*, 4th ed. New York: Lange Medical Books/McGraw-Hill

Postoperative Nausea & Vomiting (PONV)

Why do we care about PONV?

- Up to 1/3 of patients without prophylaxis will experience PONV (up to 70-80% among high-risk patients).
- Causes patient discomfort
- Prolonged PACU stay
- A leading cause of unanticipated hospital admission
- Possible aspiration risk
- Patients report avoidance of PONV as a greater concern than postoperative pain (willing to pay \$56-100 out-of-pocket for effective PONV control).

Major Risk Factors

Patient-Related

- History of PONV or motion sickness
- Female > male
- Young > old
- Non-smoker

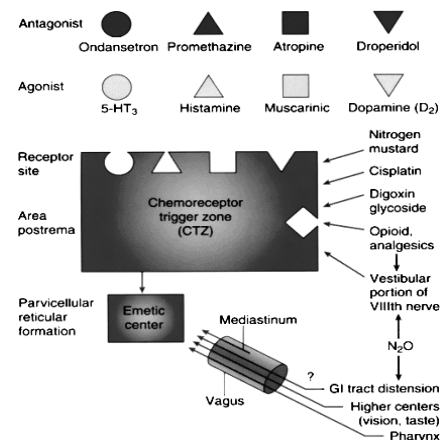
Anesthetic-Related

- N₂O, volatile anesthetics
- Drugs (narcotics, neostigmine)
- Aggressive hydration (gut edema)

Surgery-Related

- Duration of surgery - every 30 minutes increases risk by 60% above baseline (e.g. 10% → 16% after 30 minutes)
- Type of surgery (laparoscopic, ENT, neuro, breast, plastics, strabismus)

Chemoreceptor Trigger Zone



Antiemetic Classes

5-HT₃ Antagonists (e.g. Ondansetron, Granisetron)

- Serotonin receptor antagonist
- More effective at preventing emesis than nausea
- All agents equally effective
- Zofran 4-8 mg IV or Kytril 0.1-1 mg IV before end of case

Steroids

- Cheap and effective
- Can be given anytime, for prolonged PONV relief
- Avoid in diabetics
- Decadron 4-10 mg IV anytime during case

Gastrokinetic (e.g. Metoclopramide)

- Dopamine antagonist; can cause extrapyramidal SEs
- Increases GI motility and LES tone
- Reglan 20 mg IV before end of case

Antiemetic Classes

Phenothiazines (e.g. Promethazine, Prochlorperazine)

- Dopamine antagonist
- Can cause sedation and extrapyramidal side effects
- Phenergan 12.5-25 mg at end of case.

Anticholinergics (e.g. Scopolamine)

- Centrally acting
- Transdermal administration requires 2-4 hours for onset.
- Anticholinergic side effects ("mad as a hatter", "blind as a bat", "dry as a bone", "red as a beet").
- Scopolamine patch 1.5 mg TD q72hr

Butyrophenones (e.g. Droperidol, Haloperidol)

- Central dopamine antagonist
- Cheap and effective, but a "black box" warning regarding QT prolongation has caused it to fall out of favor.
- Droperidol 0.625-1.25 mg IV at end of case.

Other Antiemetic Agents

Vasopressors

- Ephedrine 50 mg IM
 - Prevents gut hypoperfusion

Induction agents

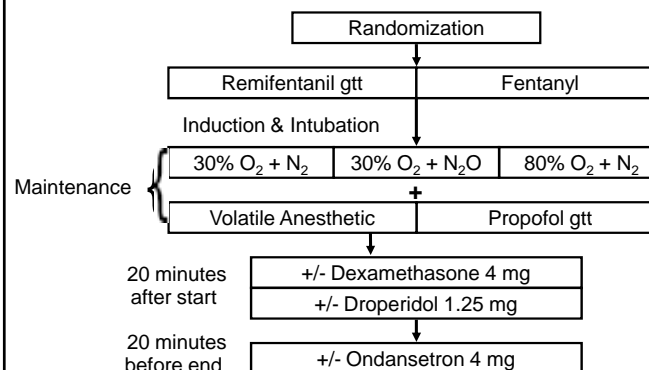
- Propofol 10-20 mg IV bolus

Antihistamines (H₂-blockers)

- Cimetidine 300 mg IV
- Ranitidine 50 mg IV

IMPACT Trial: Study Design (Apfel et al., 2004)

5161 patients, 6 treatments ($2^6 = 64$ treatment groups)

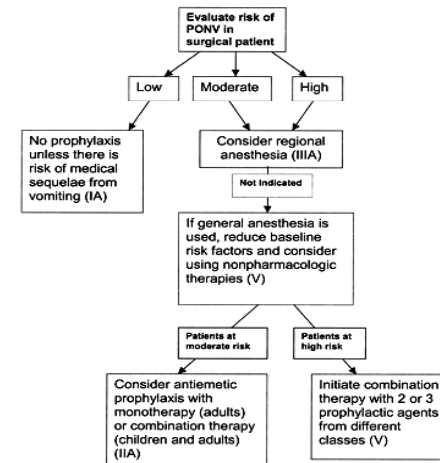


IMPACT Trial: Results (Apfel et al., 2004)

Intervention	RR Reduction	P value
Dexamethasone (vs. none)	26.4%	<0.001
Ondansetron (vs. none)	26.0%	<0.001
Droperidol (vs. none)	24.5%	<0.001
Nitrogen carrier (vs. N ₂ O)	12.1%	0.003
Propofol gtt (vs. volatiles)	18.9%	<0.001
Remifentanyl gtt (vs. fentanyl)	-5.2%	0.21

- Interventions acted independently of each other; relative risk reduction (RRR) of combined therapy can be estimated by multiplying individual RRRs.
- Average PONV = 34% (59% with volatile + N₂O + remi + no antiemetics; 17% with propofol + N₂ + fentanyl + antiemetics x3).
- Use the safest and cheapest antiemetic first; use combined therapy only in moderate or high-risk patients.

Algorithm for PONV Treatment



Strategies to Reduce PONV

- Use regional anesthesia vs. GA
- Use propofol for induction and maintenance of anesthesia
- Use intraoperative supplemental O₂ (50-80%)
- Avoid N₂O and/or volatile anesthetics
- Minimize opioids
- Minimize (<2.5 mg) or eliminate neostigmine
- Maintain euvolemia; avoid hypervolemia (gut edema)
- Use a combination of antiemetics in different classes

References

- Apfel CC, et al. 2004. A factorial trial of six interventions for the prevention of postoperative nausea and vomiting. *NEJM*, **350**: 2441-51.
- Feeley TW and Macario A. The postanesthesia care unit. In Miller RD (ed), *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005.
- Gan TJ, et al. 2001. How much are patients willing to pay to avoid postoperative nausea and vomiting? *Anesth Analg*, **92**: 393-400.
- Gan TJ, et al. 2003. Consensus guidelines for managing postoperative nausea and vomiting. *Anesth Analg*, **97**:62-71.
- Gan TJ. 2006. Risk factors for postoperative nausea and vomiting. *Anesth Analg*, **102**: 1884-98.
- Watcha MF and White PF. 1992. Postoperative nausea and vomiting: its etiology, treatment, and prevention. *Anesthesiology*, **77**: 162-84.

Extubation Criteria & Delayed Emergence

Extubation Criteria - OR

1. **Adequate Oxygenation**
 - $S_{pO_2} > 92\%$, $P_{aO_2} > 60$ mm Hg
2. **Adequate Ventilation**
 - $\dot{V}_T > 5$ ml/kg, spontaneous $RR > 7$ bpm, $ET_{CO_2} < 50$ mm Hg, $P_{aCO_2} < 60$ mm Hg
3. **Hemodynamically Stable**
4. **Full Reversal of Muscle Relaxation**
 - Sustained tetany, TOF ratio > 0.9
 - Sustained 5-second head lift or hand grasp
5. **Neurologically Intact**
 - Follows verbal commands
 - Intact cough/gag reflex

Extubation Criteria - OR

6. **Appropriate Acid-Base Status**
 - $pH > 7.25$
7. **Normal Metabolic Status**
 - Normal electrolytes
 - Normovolemic
8. **Normothermic**
 - Temp $> 35.5^\circ$
9. **Other Considerations**
 - Aspiration risk
 - Airway edema
 - Awake vs. Deep (i.e. NOT in Stage II)

Extubation Criteria - ICU

Subjective Criteria

- Underlying disease process improving.

Objective Criteria

- Adequate mentation ($GCS > 13$, minimal sedation)
- Hemodynamically stable, on minimal pressors (e.g. dopamine < 5 mcg/kg/min)
- $S_{pO_2} > 90\%$, $P_{aO_2} > 60$ mm Hg, $P_{aO_2}/F_{iO_2} > 150$ on $PEEP < 5-8$ cm H₂O and $F_{iO_2} < 0.4-0.5$
- $P_{aCO_2} < 60$ mm Hg, $pH > 7.25$

Ventilator Criteria (during SBT)

- $RSBI$ (RR/\dot{V}_T) < 100 , $NIF > 20$ cm H₂O
- $\dot{V}_T > 5$ ml/kg, $\dot{V}_C > 10$ ml/kg
- $RR < 30$ bpm

Potential Difficult Extubation

- History of difficult intubation
- OSA
- Maxillofacial trauma
- Generalized edema
- Paradoxical vocal cord motion (preexisting)
- Post-procedural complications:
 - Thyroid surgery (~4% risk of RLN injury, late hypocalcemia)
 - Diagnostic laryngoscopy +/- biopsy (laryngospasm, edema)
 - Uvulopalatoplasty (edema)
 - Carotid endarterectomy (hematoma, nerve palsies)
 - ENT surgeries (hematoma, jaw wires)
 - Cervical decompression (edema)

Approach to Difficult Extubation

- If intubation was technically difficult (e.g. multiple DLs, FOI), consider maintaining a “pathway” to the trachea (e.g. bougie, FOB, Airway Exchange Catheter).
- If airway edema is suspected due to fluids or traumatic intubation, consider performing a “Cuff-Leak Test”
 - Deflate cuff, occlude ETT, observe whether patient can breath around the tube.
 - A failed leak test does NOT always lead to failed extubation, but may warrant further patient observation; likewise, passing a leak test does NOT guarantee successful extubation.

Stages of Anesthesia

Historical terminology to describe depth of anesthesia upon gas induction. Today, more important for emergence.

Stage 1

- Sedated, intact lid reflex, follows commands

Stage 2

- Excited/disinhibited, unconscious, unable to follow commands or exhibit purposeful movement
- Irregular breathing & breath-holding, dilated & disconjugate pupils, conjunctival injection
- Increased incidence of laryngospasm, arrhythmias, and vomiting.

Stage 3

- Surgical anesthesia

Stage 4

- Medullary depression, cardiovascular/respiratory collapse

Delayed Emergence

Definition

- Failure to regain consciousness as expected within 20-30 minutes of the end of a surgical procedure.

Causes

1. Residual drug effects
 - Absolute or relative overdose
 - Potentiation of agents by prior intoxication (e.g. EtOH, illicit drugs) or medications (e.g. clonidine, antihistamines)
 - Organ dysfunction (e.g. renal, liver) interfering with metabolism/excretion.
2. Hypercapnia and/or Hypoxemia
3. Hypothermia (<33°C)
4. Hypo-/Hyperglycemia

Delayed Emergence

Causes

5. Metabolic Disturbances
 - Acid-base, hyponatremia, hypo-/hypercalcemia, hypomagnesemia
6. Organ Dysfunction
 - Renal failure, liver failure (e.g. hepatic encephalopathy)
7. Neurologic Insults
 - Seizure/post-ictal state
 - Increased ICP
8. Perioperative Stroke
 - Risk factors: AFib, hypercoagulable state, intracardiac shunt
 - Incidence: 0.1-0.4% in low-risk procedures; 2.5-5% in high-risk procedures

Diagnosis and Treatment

Ensure adequate oxygenation, ventilation, and hemodynamic stability first, then proceed with:

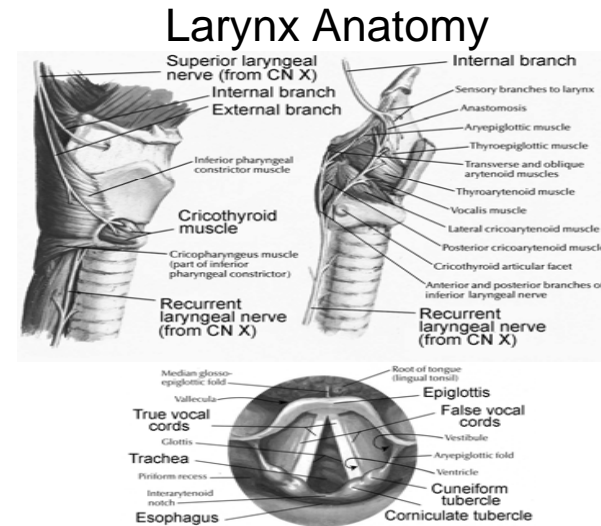
1. Administer "reversal agents"
 - Naloxone 0.40mg – 2mg IV Q 2-3 minutes. (can dilute to give in 0.04mg increments)
 - If no response after 10 mg, reconsider narcotic overdose as cause of delayed emergence
 - Flumazenil 0.2 mg IV bolus Q 45-60 seconds over 15 seconds
 - May repeat doses. Maximum of 1 mg IV bolus. No more than 3 mg total in one hour.
 - Physostigmine 1-2 mg IV (for central cholinergic syndrome)
 - Neostigmine – maximum of 5 mg IV. Give with glycopyrrolate.
2. Ensure patient is normothermic
 - Use Bair Hugger
3. Check ABG for P_aO_2 , P_aCO_2 , glucose, and electrolytes
4. Consider neurological insults
 - Perform pertinent neurologic exam
 - Consider further workup (e.g. CT, MRI, EEG)
 - Consider Neuro consult

References

- Feeley TW and Macario A. The postanesthesia care unit. In Miller RD (ed), *Miller's Anesthesia, 6th ed.* Philadelphia: Elsevier Churchill Livingstone, 2005.
- MacIntyre NR et al. 2001. Evidence-based guidelines for weaning and discontinuing ventilatory support: a collective task force facilitated by the ACCP, AARC, and the ACCCM. *Chest*, **120**: 375S-95S.
- Rashad Net University (www.rashaduniversity.com/delem.html)
- Rosenblatt WH. Airway management. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia, 5th ed.* Philadelphia: Lippincott Williams & Wilkins, 2006.

At the end of a general anesthesia case with a 60 yo male patient, I wheeled him into the PACU and he looked straight at me and very seriously said, "So, can I have your number?" His wife was in the waiting room, and I was 7 months pregnant. Classic VA.

Laryngospasm & Aspiration



Larynx Anatomy: Innervation

Nerve	Motor	Sensory
Recurrent Laryngeal (from CN X)	Thyroarytenoid (tensor) Lateral Cricoarytenoid (adductor) Transverse Arytenoid (adductor) Posterior Cricoarytenoid (abductor, tensor)	Subglottic mucosa
Superior Laryngeal (from CN X)		
• Internal branch	None	Epiglottis/BOT Supraglottic mucosa
• External branch	Cricothyroid (adductor)	Anterior subglottic mucosa

Does bilateral recurrent laryngeal nerve injury produce the same defect as succinylcholine?

Laryngospasm

Definition

- Occlusion of the glottis and laryngeal inlet by the action of the laryngeal muscles.

Predisposing Factors

- Stage 2 of anesthesia (excitement/delirium)
- Light anesthesia relative to surgical stimulation
- Mechanical irritants to the airway
 - Blood or secretions
 - Airway suctioning or instrumentation
- GERD
- Upper respiratory tract infection (0.85-5% incidence)

Laryngospasm

Prevention

- Ensure adequate anesthetic depth before manipulation
- Clear secretions before extubation
- Topicalize larynx with local anesthetic
- Muscle relaxants

Management

1. Jaw thrust, head tilt, oral or nasal airway
2. CPAP via bag-mask ventilation with 100% O₂
3. Suction oropharynx
4. Succinylcholine 10-20 mg IV, maintain airway with bag-mask or ETT until spontaneously breathing
5. Prepare for surgical airway
6. Monitor for postobstructive negative pressure pulmonary edema (NPPE)

Negative Pressure Pulmonary Edema

Causes

- Laryngospasm
- Upper airway obstruction/ETT obstruction
- Incidence of 0.1% of anesthetics

Risk Factors

- Laryngospasm
- Young (20-40 years), healthy (ASA I-II), male (80%)

Presentation

- Laryngospasm, chest wall retraction
- Frothy, serosanguinous or bloody airway secretions
- ↓S_pO₂, ↑ET_{CO2}, hypotension, large P_(A-a) gradient
- CXR with pulmonary edema

Negative Pressure Pulmonary Edema

Pathogenesis

- Negative intrathoracic pressure (up to 100 cmH₂O)
- ↑RV preload → ↑pulmonary hydrostatic pressure
- ↑RV preload → interventricular septum shift → LV diastolic dysfunction → ↑PCWP
- Hypoxia, hypercapnea, acidosis → HPV & ↑PVR
- Stress response → ↑SVR and ↑LV afterload
- Alveolar-capillary membrane leak → protein loss

Treatment

- Supportive care (O₂, IPPV, PEEP/CPAP)
- Conservative management until process reverses; consider volume and/or pressors PRN.
- Lasix is usually NOT helpful.

Pulmonary Aspiration

Predisposing Conditions

- Full stomach or unknown NPO status (e.g. trauma)
- Intra-abdominal process (bowel obstruction, ileus, inflammation)
- Gastroparesis (narcotics, DM, uremia, EtOH, infection)
- GE junction incompetence (GERD, hiatal hernia, scleroderma)
- Pregnancy, obesity
- Neuromuscular disease processes
- Difficult intubation and/or prolonged bag-mask ventilation

Pulmonary Aspiration

Prevention

- Follow NPO guidelines for routine elective cases
- Use metoclopramide, H₂-blockers, and antacids in high-risk patients
- Consider awake, regional anesthetic
- Consider awake, upright intubation and/or RSI
- If present, leave NGT to suction
- Apply cricoid pressure until ETT position confirmed
- Minimize bag-mask PPV and/or keep pressure <20 cmH₂O
- Extubate after recovery of protective reflexes

NPO Guidelines

Ingested Material	Minimum Fasting Period
Clears	2 hours
Breast Milk	4 hours
Formula	6 hours
Non-human Milk	6 hours
Light Meal	6 hours
Fatty Meal	6-8 hours

- There is no evidence for the routine use of metoclopramide, H₂-blockers, proton pump inhibitors, antiemetics, or anticholinergics in preventing aspiration or in reducing its morbidity/mortality.
- If given preoperatively, only nonparticulate antacids should be used.

Pulmonary Aspiration

Aspiration Pneumonitis

- Sterile, chemical pneumonitis caused by aspiration of acidic and particulate material
- Highest risk in patients with gastric volume >25 ml and pH <2.5.
- Aspiration does NOT always cause pneumonia!

Management

- Place patient in head-down position
- Immediately suction pharynx and trachea before PPV
- 100% O₂, intubate, apply PEEP or CPAP
- Supportive care - monitor for chemical PNA/ARDS
- Possible bronchoscopy for removal of particulate matter, if suspected
- Antibiotics are not necessary unless subsequent infection develops
- Steroids are not indicated.

References

- ASA Task Force on Preoperative Fasting. 1999. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures. *Anesthesiology*, **90**: 896-905.
- Deepika K, et al. 1997. Negative pressure pulmonary edema after acute upper airway obstruction. *J Clin Anesth*, **9**: 403-8.
- Gaba DM, Fish KJ, and Howard SK. *Crisis Management in Anesthesiology*. Philadelphia: Churchill Livingstone, Inc., 1994.
- Netter FH. *Atlas of Human Anatomy*, 4th ed. Philadelphia: WB Saunders, 2006.

Oxygen Failure in the OR

Etiology

Loss of Pipeline Oxygen

- Exhaustion of central O₂ supply.
- Obstruction of central O₂ supply line to OR.
- O₂ shutoff valve in OR is off.
- Obstruction or disconnection of O₂ hose in the OR.
- Failure of O₂ regulator in the anesthesia machine.

Faulty Oxygen Supply

- Crossing of pipelines during construction/repairs.
- Incorrect connection of gas hoses.
- Non-O₂ cylinder at the O₂ yoke.
- Wrong gas in the O₂ cylinder.
- Broken flowmeter.

Prevention

Preadesthesia Machine Check

- Check pipeline pressure ~50 psi.
- Check O₂ tanks >50% full.
- Calibrate O₂ analyzer.

Supply-Side Safety Features

- Color-coded gas tanks
- DISS, PISS, and Quick Connects

Anesthesia Machine Safety Features

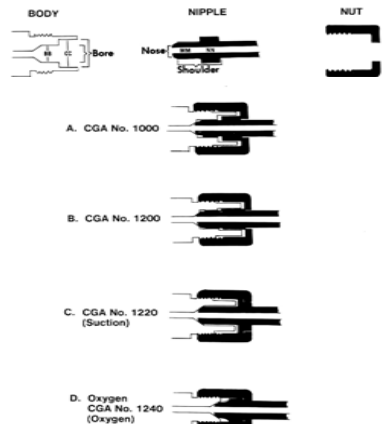
- Flowmeter arrangement
- O₂:N₂O ratio controller
- Oxygen supply failure protection device (“fail-safe valve”)

Gas Cylinders

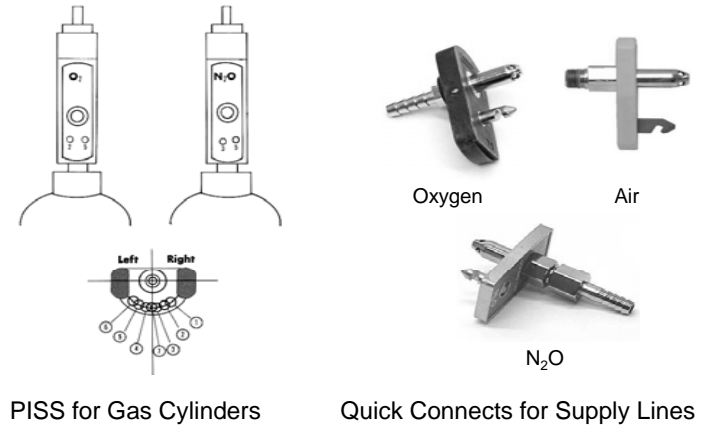
Gas	E-Cylinder Capacity (L)	Pressure (psi)	Color (USA)	Color (Int'l)	Form
O ₂	660	1900	Green	White	Gas
Air	625	1900	Yellow	White & Black	Gas
N ₂ O	1590	745	Blue	Blue	Liquid
N ₂	650	1900	Black	Black	Gas

How long can you use an O₂ tank starting at 430 psi running at 5 L/min?

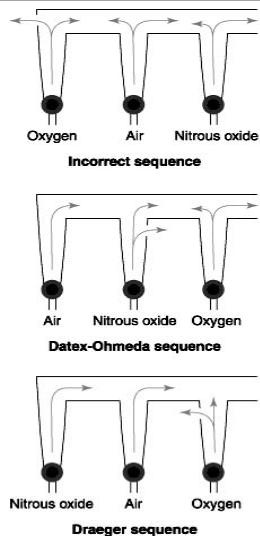
Diameter Index Safety System



Pin Index Safety System

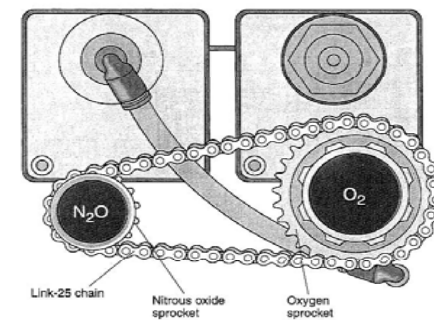


Flowmeter Arrangement



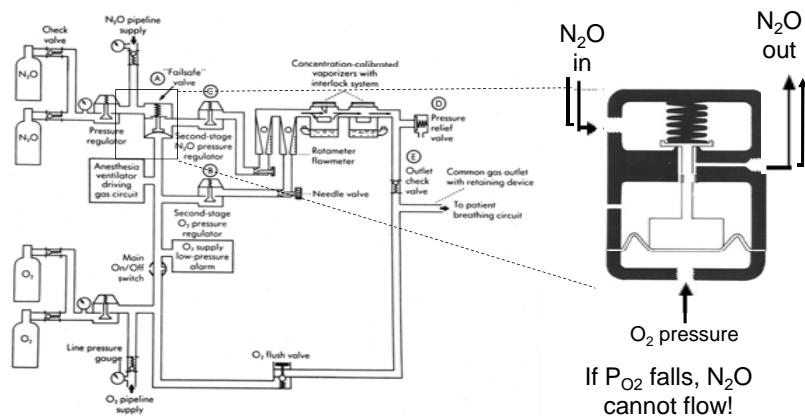
- A leak in the upstream O₂ flowmeter ("Incorrect sequence") results in a hypoxic gas mixture.
- A leak in the Datex-Ohmeda or Draeger flowmeter arrangements may deliver less Air or N₂O than expected, but the mixture will NOT be hypoxic because O₂ is closest to the FGF outlet.

O₂:N₂O Ratio Controller



Linkage mechanisms between flow valves can be either mechanical (above), pneumatic, or electronic.

Oxygen Failure Protection Device



Detection

- Pressure gauges fall (pipeline, tanks)
- Low O₂ alarms (O₂ supply failure, F_iO₂ analyzer)
- Flowmeters fall (O₂ and other gases)
- O₂ flush inoperative
- Bellows inoperative
- Apnea alarms (spirometer, capnograph)
- Increasing O₂ flow makes the problem worse
- Hypoxemia, hypercarbia
- Arrhythmias, bradycardia, cardiac arrest

Management

- Notify surgeon, call for help.
- Verify problem (pressure gauges, flowmeters, O₂ flush, O₂ analyzer, capnograph).
- Switch to O₂ cylinder (calculate remaining time).
- Use manual ventilation to conserve O₂.
- Check valves, hoses, couplers.
- D/C supply lines if crossed pipelines suspected.
- Call for backup O₂ tanks.
- Close breathing circuit, manually ventilate.
- Switch to self-inflating bag (Ambu-Bag), Jackson-Reese with external tank, or mouth-to-ETT if necessary.
- Consider switch to TIVA until cause of failure is known.

References

- Dorsch JA and Dorsch SE. *Understanding Anesthesia Equipment: Construction, Care, and Complications*, 3rd ed. Baltimore: Williams & Wilkins, 1994.
- Gaba DM, Fish KJ, and Howard SK. *Crisis Management in Anesthesiology*. Philadelphia: Churchill Livingstone, Inc., 1994.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill Companies, Inc., 2006.

Anaphylaxis

Overview

- Allergic reactions are an important cause of intraoperative morbidity and mortality (3.4% mortality)
- Account for approximately 10% of all anesthetic complications
- More than 90% of reactions occur within 3 minutes but can be delayed by hours with variable presentation
- Can be difficult to identify cause as multiple drugs are given early in anesthetic
- Usually the faster the reaction, the more severe the course will likely be
- Anaphylaxis involves a combination of systemic and dermal signs & symptoms, all due to release of vasoactive mediators which:
 - Increase mucous membrane secretions
 - Increase bronchial smooth muscle tone
 - Decrease vascular smooth muscle tone and increase capillary permeability
- Anaphylactic and anaphylactoid reactions present similarly and are treated IDENTICALLY

Anaphylaxis vs. Anaphylactoid

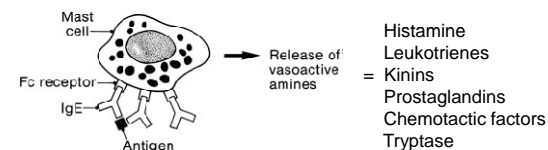
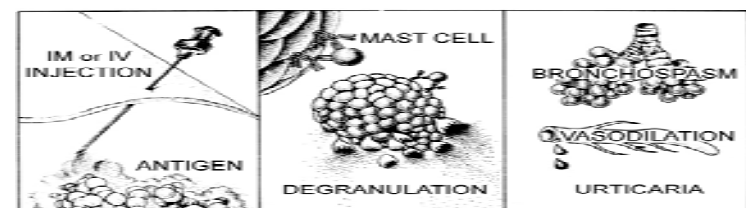
Anaphylaxis

- IgE-mediated Type I hypersensitivity reaction
- Sensitization = prior exposure to an antigen which produces antigen-specific IgE antibodies that bind to Fc receptors on mast cells and basophils.
- Upon re-exposure to the antigen, IgE antibodies then cross-link Fc receptors causing degranulation and release of stored mediators (vasoactive)
- Reaction is Dose Independent!

Anaphylactoid

- Direct activation of mast cells and basophils by non-IgE mechanisms or activation of complement system
- May occur on 1st exposure to an antigen

Sequence of Events



Common Precipitants

- Muscle relaxants (~70% responsible for anaphylaxis during GA)
 - NDMBs (rocuronium > vecuronium > cisatracurium) > Sch
- Latex (~20%): remember can occur from skin contact alone
- Antibiotics
 - Often reported ~10% (skin testing) cross-reactivity between PCN and cephalosporins; actual incidence for systemic response is now reported ~4.5%
- Local anesthetics
 - Ester > amides, possible PABA allergy.
- Propofol
 - Egg or other protein allergy (peanuts)?
- Colloids
 - Hespan (6% HES)
- Blood products
- Protamine
 - Isolated from salmon sperm, therefore patients with fish allergy or diabetics with NPH allergy have encountered reactions

Latex Allergy

- Obtain a careful history:
 - Healthcare workers
 - Children with spina bifida
 - Urogenital abnormalities (h/o multiple urogenital catheters)
 - Food allergies (mango, kiwi, avocado, passion fruit, bananas)
- Establish a latex-free environment:
 - Schedule patient as first case of the day
 - Most equipment & supplies are latex-free; if available, have a cart of latex-free alternatives available
 - Remove tops of multi-dose vials when drawing up drugs
- Prophylactic steroids and/or H1-blockers (uncertain benefit)
- Prepare for the worst, hope for the best

Sign and Symptoms

System	Symptoms (e.g. MAC/Regional)	Signs (e.g. General or Regional)
Respiratory	Dyspnea Chest tightness	Hypoxia Wheezing Laryngeal edema ↓ Compliance/↑ PIPs Pulmonary edema
Cardiovascular	Dizziness ↓ LOC	Hypotension Tachycardia Dysrhythmias Pulmonary HTN Cardiac arrest
Cutaneous	Itching	Hives Flushing Periorbital edema Perioral edema

Anaphylactic reactions may have variable presentations with some or all of these signs & symptoms.

Management

Acute Phase

1. Stop administration of offending antigen
2. Notify surgeon AND call for help
3. Maintain airway, give 100% O₂
4. In cases of severe cardiovascular collapse, would consider discontinuation of all agents that may augment hypotension such as inhaled anesthetics (via vasodilation) & narcotic infusions (via suppressing sympathetic response).
 - Give other amnestic agents (e.g. scopolamine, midazolam)
5. Fluids 2-4 L or more, as needed for hypotension
6. Epinephrine
 1. Start 5-10 mcg IV boluses, escalate as needed
 2. ACLS doses (0.1-1 mg) for cardiovascular collapse

Management

Secondary Treatment

- Intubation
- Invasive lines - large-bore IVs, arterial line, central venous catheter, Foley catheter
- Drugs
 - H1-blocker - diphenhydramine 0.5-1 mg/kg IV
 - Steroids: hydrocortisone 0.25-1 g IV, or methylprednisolone 1-2 g IV
 - Epinephrine gtt - start 50-100 ng/kg/min (4-8 mcg/min)
(Epi minidrip - 1 mg in 250 ml NS = 4 mcg/ml; run at 60 microdrips/min = 4 mcg/min; titrate to effect)
 - H2-blockers - not a first-line agent, but not harmful either!
 - Bicarbonate - 0.5-1 mEq/kg IV, as needed

Prevention

- Obtain a careful history:
 - Previous allergic reactions?
 - Atopy or asthma?
 - Food allergies?
- Test dose drugs followed by slow administration
 - reduces anaphylactoid, but not anaphylactic reactions
- Judicious use of blood products
- Use prophylactic steroids and/or H1-blockers (no clear benefit)
- Obtain consultation from an allergist if necessary.

Testing for an Allergy

- Testing may not be necessary if there is a clear temporal association between drug and reaction
- Measurement of serum mast cell tryptase levels can help establish the diagnosis in uncertain cases of anaphylaxis.
- Follow up with an allergist may be useful for establishing a diagnosis (e.g. skin testing)

References

- Gaba DM, Fish KJ, and Howard SK. *Crisis Management in Anesthesiology*. Philadelphia: Churchill Livingstone, Inc., 1994.
- Krause RS. 2006. "Anaphylaxis." eMedicine, June 13, 2006. (<http://www.emedicine.com/emerg/topic25.htm>)
- Levy JH. The allergic response. In Barash PG, Cullen BF, and Stoelting RK (eds), *Clinical Anesthesia, 5th ed*. Philadelphia: Lippincott Williams & Wilkins, 2006.
- Matthey P, Wang P, Finegan BA, Donnelly M. Rocuronium anaphylaxis and multiple neuromuscular blocking drug sensitivities. *Can J Anaesth*. 2000 Sep;47(9):890-3.
- Romano, A, Gueant-Rodriguez, RM, Viola, M, et al. Cross-reactivity and tolerability of cephalosporins in patients with immediate hypersensitivity to penicillins. *Ann Intern Med* 2004; 141:16

Advanced Cardiovascular Life Support (ACLS)

Summary of Major Changes in the 2005 Guidelines

- Emphasis on delivery of effective chest compressions.
- A single compression-to-ventilation ratio (30:2) for all single rescuers for all victims.
- Each rescue breath should be given over 1 second with visible chest rise.
- New recommendation that single shocks, followed by immediate CPR, be used for defibrillation of VF cardiac arrest.
- Rhythm checks (and pulse checks) should be performed every 2 minutes, to minimize interruptions in chest compressions.

Primary ABCD Survey

Focus: Basic CPR and Defibrillation.

- A = Airway - non-invasive techniques (head tilt-chin lift, jaw thrust, oral airway, nasal airway).
- B = Breathing - positive-pressure ventilation (bag-mask, mouth-to-mouth).
- C = Circulation - CPR until defibrillator arrives (check pulses - carotid better than femoral).
- D = Defibrillation - assess cardiac rhythm for VF/VT and provide defibrillating shock if needed.

Secondary ABCD Survey

Focus: Advanced Assessments & Invasive Therapy.

- A = Airway - ETT, LMA, Combitube.
- B = Breathing - positive-pressure ventilation via invasive airway device (hand-bag or ventilator)
- C = Circulation - CPR to circulate blood and medications.
 - Establish IV access
 - EKG for rhythm analysis
 - Vasopressors and/or antiarrhythmics as needed.
- D = Differential Diagnosis - find and treat reversible causes ("6 H's & 6 T's").

CPR Basics

- Push hard, push fast (100 compressions/min)
- Ensure full chest recoil
- Minimize interruptions in chest compressions
- One cycle = 30 compressions + 2 breaths
- Five cycles = ~2 minutes
- Avoid hyperventilation
- After the airway is secured, CPR is no longer given in "cycles." Breathing is continuous at 8-10 bpm; CPR is continuous, checking for rhythm every 2 minutes.

Differential Diagnosis

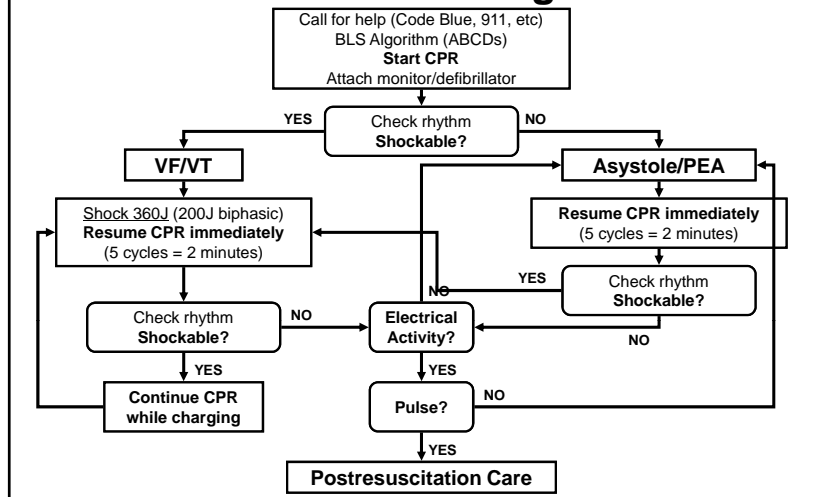
6 H's

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hyper-/Hypokalemia
- Hypoglycemia
- Hypothermia

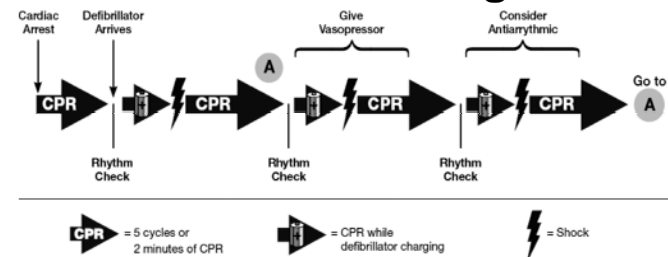
6 T's

- Toxins & Tablets
- Tamponade
- Tension pneumothorax
- Thrombosis - coronary (ACS)
- Thrombosis - pulmonary (PE)
- Trauma

Pulseless Arrest Algorithm



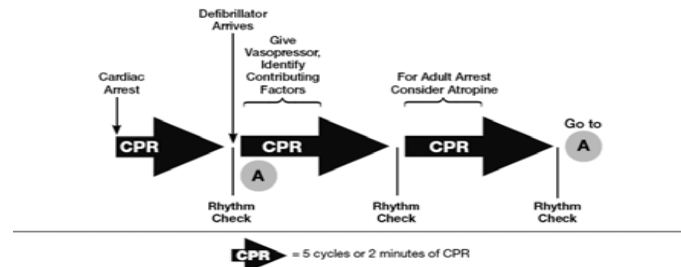
VF/Pulseless VT Algorithm



When IV access is present:

- Vasopressin 40 units x1 or Epinephrine 1 mg q3-5min
- Amiodarone 300 mg x1, may repeat 150 mg x1
- Lidocaine 1-1.5 mg/kg, then 0.5-0.75 mg/kg (max 3 mg/kg)
- Magnesium 1-2 g for torsades de pointes

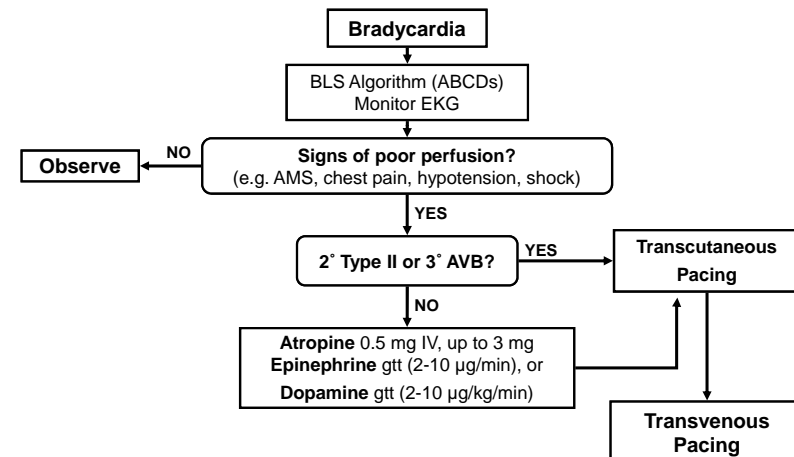
Asystole/PEA Algorithm



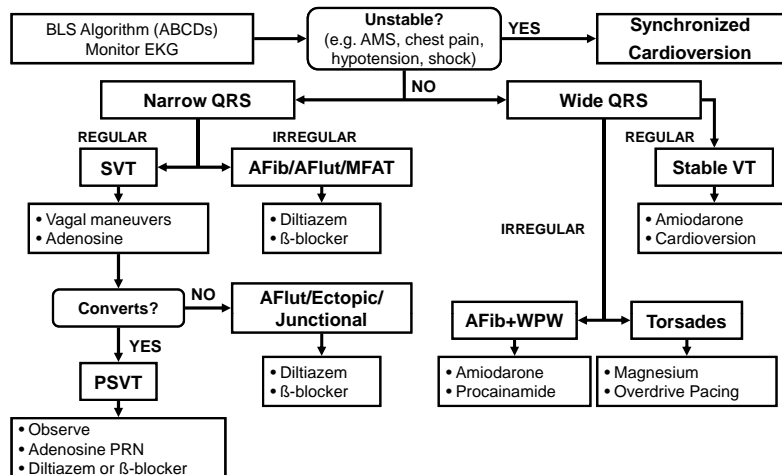
When IV access is present:

- Epinephrine 1 mg q3-5min, or Vasopressin 40 units x1
- Atropine 1 mg q3-5min (up to 3 doses)

Bradycardia Algorithm



Tachycardia Algorithm



References

- American Heart Association. 2005. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*, **112**: IV-1–IV-211.
- Field JM (ed), Hazinski MF (ed), and Gilmore D (ed). *Handbook of Emergency Cardiovascular Care: for Healthcare Providers*. American Heart Association, 2006.

Malignant Hyperthermia

Basics

Definition

- A pharmacogenetic clinical syndrome that manifests as a hypermetabolic crisis when susceptible patients are exposed to an anesthetic triggering agent.

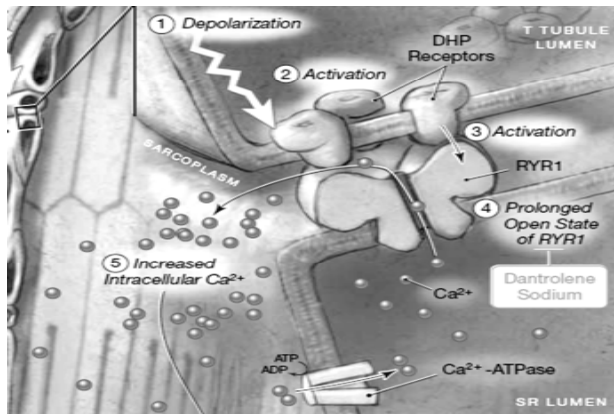
Genetics

- MH trait is found in 1:2000-3000 patients.
- Autosomal dominant with low penetrance.
- At least 4 chromosomal loci identified.
- Ryanodine receptor-1 (RYR-1) is best characterized.

Incidence

- 1 in 20,000-50,000 anesthetics, depending on the population and drugs used.
- May occur on a patient's 2nd exposure to triggers.

Excitation-Contraction Coupling



Risk Factors

- Prior history of MH
- Family history of MH
- Age (Pedi > Adult)
- History of unexplained fevers, muscle cramps, or weakness
- History of caffeine intolerance
- Trismus on induction (precedes 15-30% of MH)
- Comorbidities:
 - Central Core Disease
 - Dystrophinopathies (DMD, Becker's)
 - Other myopathies
 - King-Denborough Syndrome
- Type of Procedure:
 - Ortho (joint dislocation)
 - Ophtho (strabismus or ptosis repair)
 - ENT (cleft palate, T&A, dental procedures)

Sequence of Events

1. Triggers

- All potent inhalational agents (but not N₂O)
- Succinylcholine

2. Increased Cytoplasmic Free Ca²⁺

- Masseter muscle rigidity (trismus)
- Total body rigidity

3. Hypermetabolism

- Increased CO₂ production (most sensitive and specific sign of MH!)
- Increased O₂ consumption
- Increased heat production

Sequence of Events

4. Cell Damage

- Leakage of K⁺, myoglobin, CK

5. Compensatory Mechanisms

- Increased catecholamines - tachycardia, hypertension, cutaneous vasoconstriction
- Increased cardiac output - decreased S_{cv}O₂, decreased P_aO₂, metabolic acidosis
- Increased ventilation - increased ET_{CO2}, increased V_E
- Heat loss - sweating, cutaneous vasodilation

6. Temperature Rise

- A late and inconsistent sign of MH!
- Temperature can rise 1-2°C every 5 minutes.

Sequence of Events

7. Secondary Systemic Manifestations

- Arrhythmias
- DIC
- Hemorrhage
- Cerebral Edema
- Acute Renal Failure
- Compartment Syndrome
- Death

Differential Diagnosis

- Neuroleptic Malignant Syndrome (NMS)
- Thyroid Storm
- Sepsis
- Pheochromocytoma
- Drug-induced (e.g. ecstasy, crack, amphetamines, PCP, LSD)
- Serotonin Syndrome
- Iatrogenic Hyperthermia

Treatment (Acute Phase)

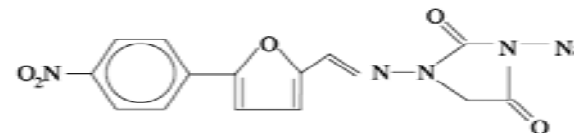
1. Get Help

- Call for help (Code Blue, 911, etc); get the MH cart.
- D/C volatile agents and succinylcholine.
- Notify surgeon; halt surgery ASAP, or continue with non-triggering agents if necessary.
- Call the MH Hotline 1-800-MH-HYPER.

2. Get Dantrolene

- 2.5 mg/kg IV push.
- Dissolve 20 mg in 60 ml sterile, preservative-free H₂O (for a 70 kg pt, you need 175 mg = 9 vials).
- Repeat until signs of MH are controlled.
- Sometimes, more than 10 mg/kg is necessary (= 35 vials of dantrolene!).

Dantrolene



- A hydrophobic, hydantoin derivative
- Interferes with excitation-contraction coupling by binding the RYR-1 Ca²⁺ channel
- Relatively safe drug; causes generalized muscle weakness (including respiratory muscles).
- Can also be used to treat NMS or thyroid storm.

Treatment (Acute Phase)

3. Treat acidosis

- Hyperventilate patient with 100% O₂ at > 10 L/min.
- Bicarbonate 1-2 mEq/kg until ABG available.

4. Treat hyperthermia

- Cool if T > 39°C, but D/C if T < 38°C.
- Apply ice to body surfaces.
- Cold NS via IV.
- Lavage stomach, bladder, or rectum PRN.

5. Treat dysrhythmias

- Standard therapies, but avoid CCBs in the presence of dantrolene (may promote hyperkalemia).

Treatment (Acute Phase)

6. Treat hyperkalemia

- Hyperventilate
- Bicarbonate
- Insulin & glucose (10 units in 50 ml D50)
- Calcium (10 mg/kg CaCl₂, or 10-50 mg/kg Ca gluconate)

7. Maintain UOP

- Mannitol (0.25 g/kg), and/or
- Lasix (1 mg/kg)

8. Continue to monitor

- ET_{CO2}, Temp, UOP & color, Electrolytes, ABG, CK, PT/PTT/INR

Treatment (Post Acute Phase)

1. **Observe in ICU for at least 24 hours.**
 - Recrudescence rate is 25%.
2. **Dantrolene**
 - 1 mg/kg IV q4-6hrs for at least 36 hours.
3. **Follow labs**
 - ABGs, CK, myoglobinuria, coags, electrolytes, UOP and color
4. **Counsel patient and family**
 - Future precautions.
 - Refer to MHAUS.
5. **Refer patient and family to nearest Biopsy Center for follow-up.**

Susceptibility Testing

Caffeine-Halothane Contracture Test (CHCT)

- Gold Standard
- Requires fresh muscle biopsy
- Sensitivity >97%, Specificity 80-93%
- Available at 9 U.S. testing centers

Molecular Genetics

- *RYR1* mutation screening
- Low sensitivity, but high specificity
- Typically reserved for patients with a positive CHCT, relatives of known MH susceptibility, or patients with highly suspicious MH episode.

Prevention

Machine

- Change circuit and CO₂ absorbant
- Remove vaporizers
- Flush machine at FGF of 10 L/min for 20 minutes.

Monitors

- ASA monitors, especially temperature and ET_{CO2}

Anesthetic

- Avoid succinylcholine and volatiles
- All other non-triggering agents are OK (including N₂O)

Emergency

- Know where to find the MH cart.
- Have dantrolene available.

References

- Litman RS, Rosenberg H. 2005. Malignant hyperthermia: update on susceptibility testing. *JAMA*, **293**: 2918-24.
- Morgan GE, Mikhail MS, and Murray MJ. *Clinical Anesthesiology, 4th ed.* New York: McGraw-Hill Companies, Inc., 2006.
- Malignant Hyperthermia Association of the United States (MHAUS, <http://www.mhaus.org>)
- UCLA Department of Anesthesiology (<http://www.anes.ucla.edu/dept/mh.html>)

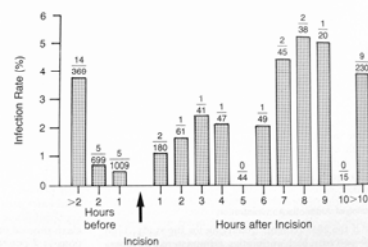


Perioperative Antibiotics

Timing of prophylaxis

- Antibiotic therapy should be given within 60min prior to surgical incision for adequate serum drug tissue levels at incision.
- If vancomycin or a fluoroquinolone is used, it should be given within 120 min of incision to prevent antibiotic-associated reactions around the time of anesthesia induction.
- If a proximal tourniquet is used, the entire antibiotic dose should be administered before the tourniquet is inflated.

Timing of prophylaxis



- Rates of Surgical-Wound Infection Corresponding to the Temporal Relation between Antibiotic Administration and the Start of Surgery
- The number of infections and the number of patients for each hourly interval appear as the numerator and denominator, respectively, of the fraction for that interval. The trend toward higher rates of infection for each hour that antibiotic administration was delayed after the surgical incision was significant (z score = 2.00; $P < 0.05$ by the Wilcoxon test).

Classen DC, et al. (1992) The timing of prophylactic administration of antibiotics and the risk of surgical-wound infection. *The New England Journal of Medicine* 326:281-286.

Administration and Redose

- To be given via slow infusion (over 1 hour; reconstitute in 100ml NS)
 - Vancomycin (Red Man Syndrome)
 - Clindamycin (QT prolongation)
 - Gentamicin (ototoxicity)
 - Metronidazole
 - Typical dosages for antibiotics commonly used in the OR:
 - Ampicillin 1gm
 - Cefazolin 1-2gm (consider 2gm for patients > 80kg)
 - Cefoxitin 1-2gm
 - Clindamycin* 600mg
 - Gentamicin* 1.5mg/kg
 - Metronidazole 500mg
 - Zosyn 3.375gm
 - Ceftriaxone 1gm
 - Vancomycin 1gm
 - * can potentiate neuromuscular blockers
 - Consider re-dosing every 6 hrs (except Vanc, Zosyn, and Ceftriaxone)
 - Adjust for renal insufficiency (except for Clindamycin and Ceftriaxone)
- Note: Ertapenem is favored by Drs. Shelton and Rhoades for their colorectal cases (reconstitute 1gm in 100ml NS, infuse over 30min)

Types of Procedures

- Clean procedures (e.g. ortho, breast)
 - 1st generation cephalosporin (Cefazolin 1-2g IV), covers staphylococci and streptococci
- Procedures involving bowel anaerobes, Gram neg-bacilli, enterococci
 - 2nd generation cephalosporin (Cefoxitin 1-2g IV or Cefotetan)
 - Bowel aerobic gram-neg bacilli (e.g. E. coli) can be resistant, so consider adding metronidazole 500mg IV.
- Craniotomies-
 - 3rd generation cephalosporin, good CSF penetration (Ceftriaxone 1-2g IV)
- If pt is having colorectal surgery, hysterectomy, or vascular surgery involving a groin incision, can add gentamicin, ciprofloxacin, levofloxacin, or aztreonam to cover gram-neg bacteria.

Allergies and Interactions

- Penicillins and cephalosporins have similar β -lactam ring
- True incidence of allergy in patients with a history of PCN allergy is less than 10%. Only IgE-mediated reaction (type I, immediate hypersensitivity reactions) are true allergic reactions.
- The cross-reaction rate between PCN and cephalosporins is substantially less than 10%
- History of PCN allergy is a general risk factor for allergic manifestations to antibiotic administration that may not be specific to cephalosporins
- Cross-reaction rate between 3rd generation cephalosporins and PCN approaches 0% !
- For PCN-allergic patients, consider vancomycin or Clindamycin \pm one of the following for Gram neg coverage (ciprofloxacin, levofloxacin, gentamicin, or aztreonam)

Allergies and Interactions

- If the allergic reaction to PCN is only “rash” or “hives,” many attendings would give a cephalosporin, but always ask your specific attending!
- However, if the allergic reaction to PCN is anaphylaxis, that’s an absolute contraindication to cephalosporins.
- **Test dose:** Not always done. However, it may be prudent to give 1ml of the antibiotic first to see if the patient will have a reaction. This test dose only decreases the *anaphylactoid* reaction, not anaphylaxis.
- Allergic reactions are more likely from neuromuscular blockers than antibiotics.

Special considerations

- The American Heart Association guidelines recommend prophylaxis for those with conditions that place them at increased risk for infective endocarditis AND for those at highest risk for adverse outcomes when endocarditis does occur. These are patients with:
 - Prosthetic cardiac valve
 - Previous history of infective endocarditis
 - Congenital heart disease and completely repaired congenital heart defect if it’s within the first 6 months.
 - Cardiac transplant patients who develop cardiac valvulopathy
- Bacterial Endocarditis prophylaxis
 - Ampicillin 1-2gm IV, 30min prior to surgery and
 - Gentamicin 1.5mg/kg IV, 30min prior to surgery
 - IF PCN allergic, use Cefazolin or ceftriaxone 1gm IV, or Clindamycin 600mg IV
- For mitral valve prolapse, do not need prophylaxis because while there is increased risk for IE, the most serious adverse outcomes of IE do not usually occur in patients with this condition.
- Do not need prophylaxis for bronchoscopy without biopsy, vaginal delivery, hysterectomy, or GI/GU procedures, including colonoscopy.

References

- American Society of Anesthesiologists, ACE Program 2008. Pages 44-47.
- Ann S, Reisman RE. Risk of administering cephalosporin antibiotics to patients with histories of penicillin allergy. *Ann Allergy Asthma Immunol* 1995; 74:167-170
- Antimicrobial prophylaxis for surgery. *Treat Guidel Med Lett* 2009; 7:47
- Bratzler, DW, Hunt, DR. The surgical infection prevention and surgical care improvement projects: national initiatives to improve outcomes for patients having surgery. *Clin Infect Dis* 2006; 43:322
- Bratzler, DW, Houck, PM. Antimicrobial prophylaxis for surgery: an advisory statement from the National Surgical Infection Prevention Project. *Clin Infect Dis* 2004; 38:1706
- Classen DC, et. Al. The timing of prophylactic administration of antibiotics and the risk of surgical-wound infection. *The New England Journal of Medicine* 1992; 326:281-286.
- Pinichero ME. Use of selected cephalosporins in penicillin-allergic patients: a paradigm shift. *Diagnostic Microbiology and Infectious Disease* 2007; 57:13-18.

I met my next patient in the VA preop area. I did my physical exam and was ready to place the IV. I had the lidocaine needle at his skin and announced, "Small prick!" He responded, "Honey, that's what my ex-wife used to tell me, too."

It was time to bring the patient to the OR, and I was pushing him on a gurney down the ASC hallway. I got lost along the way and took a wrong turn leading to a dead end. I tried to play it off that we had taken this round about way just to get a patient hat for the OR. Unfortunately, despite the Versed, I think he saw right through the subterfuge.

Wheeled the patient into the room for a hip fracture repair. Nurse on the computer. Myself, anesthesia attending and ortho resident move the patient to the OR bed at which point the pt chuckles and smiles. I ask "what's so funny?" He responds, " I just had about a million dollars worth of education move me from one bed to another."

OR Setup MS MAIDS

Machine checked

High flow O2 present
Ambu Bag present

Suction on

with appropriate catheter

Monitors on patient

current vital signs displayed
know patient's baseline values
NIBP to q1 minute

Airway equipment

intubating equipment ready
other planned airway supplies ready

IV Access

runs freely into patient
adequate amount of fluid in bag

Drugs

induction agent
paralytic
narcotics
emergency drugs

Special

extra equipment needed for this case

Typical Drugs

	Typical Concentration	Dosing	Typical Dose for 70 kg Adult
<i>Anxiolytic – Preop</i> Midazolam	1 mg/mL in 2 mL vial		1-2 mg
<i>Opiod Analgesic</i> Fentanyl	50 mcg/mL in 5 mL vial	intra-op analgesia intubation	25-50 mcg 150-250 mcg
<i>Induction Agents</i> Propofol	10 mg/mL in 20 mL vial	1-2 mg/kg	150 mg
Etomidate	2 mg/mL in 20 mL vial	0.3 mg/kg	20 mg
Thiopental	25 mg/mL in 20 mL vial	3-5 mg/kg	200 mg
<i>Muscle relaxants</i> Succinylcholine	20 mg/mL in 10 mL vial	1-2 mg/kg	100 mg
Vecuronium	mix to 1 mg/mL	0.1 mg/kg	7 mg
Rocuronium	10 mg/mL in 5-10 mL vial	0.6 mg/kg RSI 1 mg/kg	40 mg 70 mg
Cisatracurium	2 mg/mL in 10 mL vial	0.15 mg/kg	10 mg
<i>Reversal agents (always give glycopyrrolate first or mixed in same syringe)</i>			
Glycopyrrolate	0.2 mg/mL in 1 mL vial	0.008-0.01 mg/kg	0.6 mg
Neostigmine	0.5 mg/mL in 10 mL vial	0.03-0.05 mg/kg	3 mg (MAX 5 mg)
<i>Pressors</i> Ephedrine	mix to 5 mg/mL		5-10 mg
Phenylephrine	mix to 100 mcg/mL		50-100 mcg
Epinephrine	mix to 10 mcg/mL		5-10 mcg



Typical Drugs Cognitive Aid
v3.0 - 5/17/2010



Hypotension

Page 1
of 2

Immediate Lifesaving Actions

Feel for **Pulse**

Check **Heart rate**: If Slow, Treat

Check **Rhythm**: if Abnormal, go to ACLS Protocol

Call for **Help**

Inform **Surgeon** and **Team**

Inspect **Surgical Field** for Blood Loss or Manipulation

Give **IV Fluid** Bolus

Give **Phenylephrine** or **Ephedrine** to Temporize

Consider **Trendelenburg** or Elevation of Patient's Legs

Turn Down or Off **Anesthetic Agent**

Consider **100% O₂**

Rule Out **Pneumothorax**: Listen to Breath Sounds

Rule Out **Auto-PEEP**: Disconnect and Reconnect Circuit

If Refractory Severe Hypotension,

Consider Code Cart

Discuss Pausing Surgery

Call for more Help

Consider Epinephrine or Vasopressin



Hypotension Cognitive Aid
v3.0 - 5/17/2010



Hypotension

Page 2
of 2

Secondary Actions

More **IV access**

Call for **Rapid Infuser**

Call for **Blood**

Place **Arterial line**

Send **Labs**: ABG, Hgb, lytes, Calcium, Type & Cross

Consider **terminating surgical procedure** or get surgical help

Consider **Trans-Esophageal Echo (TEE)** if unclear cause

Foley catheter if not present

Consider **Hydrocortisone**

Differential Diagnosis

MAP=CO x SVR; CO=SV x HR

SV from preload, afterload, contractility

Decreased preload: Hypovolemia from bleeding or other decreased volume, Tamponade, pneumothorax, PEEP, surgical compression/retraction, insufflations, pulmonary embolus, tachycardia or arrhythmia

Low SVR (vasodilation): Anaphylaxis, Cement/emboli, Anesthetics and drugs (volatiles, induction agents, Ace Inhibitors), Sepsis, Neuraxial blockade, Spinal shock

Decreased Contractility: Low Calcium, Cardiomyopathy, MI/ischemia, prolonged hypoxemia, valvular disease

Low HR if on Beta Blockers, may not get tachycardic compensation for low SV

Increased afterload: Heart unable to eject enough blood against high afterload

Low Stroke Volume: See preload, contractility, and afterload

Hypoxemia

Page 1
of 2

If Low O_2 Sat, paO_2 , or blue pt...

Immediate Lifesaving Actions

- **100% O_2 with high flows**
- Check gas analyzer to **rule out Low FiO_2 or High N_2O**
If suspect issues, ventilate with room air and see Anesthesia Machine/ O_2 Failure
- **Check other vitals** (cycle NIBP) and PIP
- **Check for $ETCO_2$** (? Extubated, disconnected, low BP)
- **Check surgical field and feel for pulse**
- Handbag to **check compliance/leaks**
and decrease machine factors
- **Listen for Breath Sounds** (Bilateral? Clear?)
- **Soft suction via ETT** (to clear secretions, check obstructions)
- **Call for HELP**, especially if no clear cause or worsening
- **Consider Code Cart** if severe
- **Consider artifact last** (only if all else ok);
switch pulse ox location or machine
- **Communicate problem to surgeon and team**

Hypoxemia

Page 2
of 2

Follow Up Actions - Consider

Nebulizers to bronchodilate - ABG - CXR

Fiberoptic to **confirm ETT position** and check for mucus plugging

If intubated and fighting ventilator, **consider additional neuromuscular blockade**

Large recruitment breaths by hand bag (if ? atelectasis and pt not hypotensive)

Artifact? Check waveform, probe position (light over fingernail), cover to prevent ambient light, ask to stop cautery, check if dyes used, move probe to ear or different site, check abg if still unclear

Consider terminating surgery if not improving

Plan for Postop care: Stay intubated? ICU? Full report to surgical and postop teams

Differential Diagnosis

Hypoventilation Check for: circuit leaks, low TV/RR or MV, residual nmb, high $ETCO_2$, high PIP, kinked or obstructed ETT, poor chest rise, etc

Low FiO_2 If low FiO_2 on '100% O_2 ' go immediately to alternate source: TANKS (back of machine: must open valve and disconnect wall hoses to activate tank flow; Call for O_2 tank with regulator - use with ambu; If low FiO_2 note auxiliary O_2 source on machine is dangerous too because same source! If <21% try room air with ambu while await new source

V/Q Mismatch or Shunt e.g. Mainstem intubation, bronchospasm (asthma/Reactive Airway Disease, ?anaphylaxis), Pneumothorax, embolus (air, blood, fat, AFE), atelectasis, aspiration, Pneumonia, mucus plug, pleural effusion, any cause hypotension with poor perfusion (e.g. hypovolemia, MI, tamponade, sepsis, etc.)

Diffusion problem usually chronic

Artifacts e.g. poor waveform (probe malposition, cold extremity, light interference, cautery), dyes (methylene blue, indigo carmine, blue nail polish)

Anesthesia Machine or O2 Failure

Immediate Lifesaving Actions

Disconnect the patient from the machine and ventilate with an Ambu™ bag on room air

Do NOT connect the patient to auxiliary flowmeter on machine – comes from SAME central source

Obtain full E cylinder of OXYGEN with a regulator
OR

Disconnect pipeline oxygen and open O2 tank on back of anesthesia machine (check not empty)

Connect Ambu™ bag or Jackson Rees circuit to oxygen tank and ventilate

Connect adaptor to allow monitoring of respiratory gases - *Is the patient receiving 100% oxygen?*

Call for help & Diagnose machine problem

Maintain anesthesia (if necessary) with IV drugs



CPR



Call for Help & Crash Cart



100 compressions/minute



2 minutes continuous CPR

Check

Always ...

- Backboard
- Establish airway
- IV Access

In the OR ...

- Turn OFF Volatile
- 100% O₂
- Check vent rate (6 breaths/minute)

Treat

Epinephrine - 1 mg IV push q 3-5 minutes

If rate slow: **Atropine** - 1 mg IV q 3-5 minutes

Consider: **Vasopressin** - 40 units

Diagnose

Find & Treat Cause - more details on back:

- | | |
|--------------------------------|------------------------|
| ① Hypovolemia | ⑦ Toxins (overdose) |
| ② Hypoxia | ⑧ Tamponade - cardiac |
| ③ Hydrogen ion - acidosis | ⑨ Tension pneumothorax |
| ④ Hyper- or Hypokalemia | ⑩ Thrombosis coronary |
| ⑤ Hypoglycemia or Hypocalcemia | ⑪ Thrombosis pulmonary |
| ⑥ Hypo- or Hyperthermia | |



Find & Treat Cause - H & Ts

- ① **Hypovolemia** - Administer rapid bolus of IV fluid and check hemoglobin/hematocrit. Give blood for anemia or massive hemorrhage.
- ② **Hypoxia** - 100% FiO₂. Confirm oxygen connections. Check for bilateral breath sounds. Suction ET tube Reconfirm ET tube placement. Consider chest x-ray.
- ③ **Hydrogen ion (acidosis)** - Check blood gas for acidosis. Administer sodium bicarbonate. Consider increasing ventilation rate but realize this will decrease effectiveness of CPR.

- ④ **Hyperkalemia** - Check blood gas for electrolyte abnormalities. Give sodium bicarbonate; glucose + insulin; calcium chloride; possibly albuterol.

Hypokalemia - Rapid but controlled infusion of potassium & magnesium

- ⑤ **Hypoglycemia or Hypocalcemia** - Check blood gas or finger stick.
- ⑥ **Hypothermia** - Active warming by forced air blanket, warm IV. Consider cardiopulmonary bypass.

Hyperthermia - Cool with axillary ice packs, cold IV. Consider peritoneal lavage. If anesthetic exposure, consider Malignant Hyperthermia. Call for MH Cart. Treat with Dantrolene. MH Hotline 800-644-9737 (MH-Hyper)

- ⑦ **Toxins** - Consider overdose of medication. Confirm no infusions are running. Confirm volatile anesthetic off.
- ⑧ **Tamponade (Cardiac)** - Consider placing transesophageal (TEE) or transthoracic (TTE) echo to rule out. Treat with pericardiocentesis.
- ⑨ **Tension Pneumothorax** - Unilateral breath sounds, possibly distended neck veins and deviated trachea (late signs). Perform emergent needle decompression followed by chest tube placement. Call for chest x-ray, but do not delay treatment.
- ⑩ **Thrombosis (Myocardial Infarction)** - Consider using TEE to evaluate wall motion of ventricle. Consider emergent coronary revascularization or fibrinolytic agents.
- ⑪ **Thrombosis (Pulmonary Embolus)** - Consider TEE to evaluate right ventricle. Consider fibrinolytic agents.

VTach VFib

CPR



Call for **Help & Crash Cart**



100 compressions/minute



2 minutes continuous **CPR**

Check

Always ...

- Backboard
- Establish airway
- IV Access

In the OR ...

- Turn OFF Volatile
- 100% O₂
- Check vent rate (6 breaths/minute)

Treat

DEFIBRILLATE - 200 Joules (Biphasic)

Resume CPR Immediately

Epinephrine - 1 mg IV push q 3-5 minutes
OR **Vasopressin** - 40 units IV push once

Consider

Consider Antiarrhythmics: **Amiodarone** 300 mg IV

Lidocaine 100 mg IV

if hypoMg or Torsades, **Magnesium Sulfate** 2 grams IV

if hyperK, **Calcium, Insulin & Glucose, Sodium Bicarbonate**

Repeat Cycle of CPR, Defib, & Meds

Symptomatic Bradycardia

Immediate Lifesaving Actions

Pulse?

If NO pulse, go to PEA Algorithm

If pulse present but hypotensive, proceed...

- Use **100% O₂**
- Confirm adequate **ventilation** and **oxygenation**
- Consider turning down or OFF all **anesthetics**

Meds

Atropine 0.4 to 1.0 mg IV

Dopamine 5 to 20 mg / kg / min

Epinephrine 10 mcg to 1 mg IV push q 3-5 minutes

Isoproterenol 2 to 10 mcg / kg / min

Secondary Actions

Place **Arterial Line**

Send **Labs**; ABG, Hemoglobin, Electrolytes

Rule Out **Ischemia**: Consider EKG, Troponins

Consider Transcutaneous **Pacing**

- set rate to at least 80 bpm
- increase current until capture achieved
- confirm patient has pulse with capture

Peds Anesthesia

Page 1
of 2

AGE	Term	6 mo	1 yr	2 yr	5 yr	10 yr
kg	3.5	6	8	13	20	40
lbs	7.5	12	17	28	42	80
ETT size	3	3.5	4	4.5	5	cuffed
ETT depth	9	10.5	12	13.5	15	18
Blade	mil 0	mil 1	mil 1	mil 1.5 mac 1	mil 1.5 mac 2	mil 2 mac 2-3
Oral Airway	0 50 mm	0 50 mm	1 50 mm	2 70mm	3 80mm	3 80mm
LMA	1	1	1.5	2	2	2.5-3.0
IV bolus	35	60	80	130	200	400
CVP depth	4-5 cm	6 cm	7 cm	9 cm	10 cm	12 cm
foley	6	8	8-10	8-10	10	12

ETT size = (age/4) + 4

ETT depth = ETT size * 3

Peds Scheduler	723-3176
Peds Emergency	721-6696
LPCH OR Front desk	721-2820
Anes Tech- OR / Out of OR	725-0034
Pre-Op Intake / Holding	721-6562 / 721-6562
PACU	494-3451
Pharmacy OR / Main LPCH	721-2731
Peds Pain Service	497-8057/pgr 18779



Peds Anesthesia

Page 2
of 2

INDUCTION Drugs

Midazolam	0.5 mg/kg PO	0.03 mg/kg IV
Propofol	2-3 mg/kg IV	
Ketamine	0.5-2 mg/kg IV	3-5 mg/kg IM
Rocuronium	0.6-1.2 mg/kg IV	
Succinylcholine	1-2 mg/kg IV	2-4 mg/kg IM
Cefazolin	25 mg/kg q6 IV	(max 1 g)
Glycopyrrolate	5-10 mCg/kg IV	
Neostigmine	0.07 mg/kg IV	

Acetaminophen	10-15 mg/kg PO	30 mg/kg PR
Fentanyl	0.5-1 mCg/kg IV	1-2 mCg/kg IM
Hydromorphone	10-20 mCg/kg IV	
Morphine	0.1 mg/kg IV	

RESUCITATION drugs (CPR doses)

Defibrillation	2 joules/kg, 2-4 j/kg, then 4 j/kg
Cardioversion	0.5-1.0 sync joules/kg
ETT drugs (LEAN)	Lido, Epi, Atropine, Narcan
Epi (CPR dose)	0.01 mg/kg IV or ETT
Atropine	0.02 mg/kg IV (min. 0.1 mg)
Naloxone	0.1-10 mCg/kg IV
Racemic Epi nebs	0.5 ml (of 2.25%) in 2.5 ml NS
Albuterol nebs	0.2-0.3 ml in 2.5 ml NS
Benadryl	0.125-0.25 mg/kg IV
Dexamethasone	0.3-1 mg/kg IV

